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DEC 18 1935

VOLUME 25 NUMBER 12

DECEMBER, 1935

Phytopathology

An International Journal

Official Organ of
The American Phytopathological Society

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Price { $6.00 per year, United States and dependencies,
$6.25 per year, Canada
$6.50 per year, elsewhere

PUBLISHED MONTHLY BY THE SOCIETY

OFFICE OF PUBLICATION

COR. LIME AND GREEN STREETS, LANCASTER, PA.

Entered at the post-office in Lancaster, Pa., as second-class matter.

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SCIENCE PRESS, Lime and Green Sts., Lancaster, Pa.	PRINTERS OF SCIENTIFIC JOURNALS, BOOKS AND MONOGRAPHS.
TRIARCH BOTANICAL PRODUCTS, Geo. H. Conant, Ripon, Wis.	PRESERVED PLANTS. MICROSCOPE SLIDES.
PHYTOPATHOLOGICAL CLASSICS, H. H. Whetzel Mgr., Cornell University, Ithaca, N. Y.	NUMBERS 1, 2, 3, AND 4, PHYTOPATHOLOGICAL CLASSICS.
THE H. W. WILSON CO., 950–972 University Ave., New York, New York.	THE GENERA OF FUNGI F. E. CLEMENTS AND C. L. SHEAR.

PHYTOPATHOLOGY

An International Journal
Official Organ of The American Phytopathological Society

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Advertisements. Rates may be secured from business management. No indorsement of any statement of claims made in advertisements is assumed by this journal or by The American Phytopathological Society.

Requests to supply copies of the journal must be made within 60 days from date of issue. Date of issue January 15 and monthly thereafter.

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PHYTOPATHOLOGY

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THE EFFECT OF LIGHT INTENSITY AND TEMPERATURE ON INFECTION OF HOPE WHEAT BY PUCCINIA GRAMINIS TRITICI[1]

HELEN HART[2] AND KAROL ZALESKI[3]

(Accepted for publication February 26, 1935)

There are many puzzling discrepancies in the degree of infection caused by *Puccinia graminis* on cereals in different developmental stages. Seedlings of Hope wheat are susceptible to *Puccinia graminis tritici* form 21 when inoculated artificially in the greenhouse. In the field throughout the United States, however, previous to 1933 Hope seldom had more than a trace of stem rust; and whenever rust did appear there was evidence of considerable resistance on the part of the host.

Stakman (30) and Stakman and Piemeisel (33) early observed that cereals and wild grasses varied greatly in their susceptibility to stem rust; some apparently were extremely susceptible when young and much less susceptible when older, others appeared to be more susceptible as they grew older, and many were susceptible in the greenhouse but not severely or universally rusted in the field. Stakman and Piemeisel (33) observed no cases of rust immunity due to age of host, although the rust often was distinctly more virulent on younger or older plants, depending on the peculiarity of the grasses. Levine (21) was of the opinion, from observational evidence, that susceptibility of plants depends to a considerable extent on their stage

[1] Published with the approval of the Director as paper No. 1324 of the Minnesota Agricultural Experiment Station.

This work was started as a cooperative project of the Division of Cereal Crops and Diseases, Bureau of Plant Industry, United States Department of Agriculture and the Minnesota Agricultural Experiment Station.

[2] Assistant Plant Pathologist, Minnesota Agricultural Experiment Station and formerly Agent in the Division of Cereal Crops and Diseases, Bureau of Plant Industry, United States Department of Agriculture.

[3] Adjoint in Botany and Plant Pathology, University of Poznan, and Fellow on the Fund of National Culture under the President of the Council of Ministers at Warsaw, Poland, in 1930 and 1931, stationed at the Minnesota Agricultural Experiment Station January to April, 1931. The junior author wishes to express his deepest thanks to the Minnesota Agricultural Experiment Station, to the United States Department of Agriculture, and to the senior author for his opportunity to assist in the greenhouse and laboratory work during the short time he was at Saint Paul, Minnesota.

of development, susceptibility being greater in the seedling and heading stages and less in the jointing stage, so that some varieties appear to be susceptible as seedlings but resistant as they mature in the field. In some wheat varieties such field resistance has been merely an escape from rust by reason of early ripening. Other varieties have appeared resistant in the field because physiologic forms to which their seedlings are susceptible have not been present or have not been abundant enough to cause wide-spread infection. Still others are rust resistant in the field because their stomata remain closed at critical periods for infection and exclude the rust germ tubes. At first it was believed that Hope wheat had a functional resistance by reason of its stomatal behavior (18), particularly since Abbott (1, 2) reported Hope completely susceptible to stem rust in the field in Peru. In 1931 and 1932, however, stomatal movements in this variety grown at St. Paul, Minnesota, were such that stem rust germ tubes might enter the host easily; but even so there was merely a trace of rust in the plots supplied with abundant inoculum of *P. graminis tritici* 21.

Other workers have advanced the theory of mature plant resistance, implying that as the host plant grows older, it acquires more resistance to rust than it had in its seedling stage. Goulden, Newton, and Brown (16) tested the reactions of twelve wheat varieties and two emmers at two stages of maturity, the seedling stage and the after-heading stage, to a number of physiologic forms of *Puccinia graminis tritici*. They concluded that Acme, Hope, Pentad, and H–44–24 developed marked mature-plant resistance, whereas Garnet, Marquis, and Quality did not, for the rust reactions of these three in the after-heading stages corresponded very closely with their reactions as seedlings. The authors state that the reading of reactions was difficult because there was extreme variation on different plants; consequently the readings were made on the basis of pustule size, not so much attention being paid to chlorosis and necrosis.

Harrington and Smith (17), on the other hand, studied rust reactions in seedlings and in headed plants of Marquillo, Marquis, Iumillo, and Vernal emmer. They found a strong relationship between the seedling reaction and the after-heading reaction, although the percentages of infection after heading often were low as compared with those in seedlings.

Neatby (24), in his study of Marquillo × Reward, Garnet × Marquillo, and Garnet × Double Cross, found no evidence of mature plant resistance but indications that "the inheritance of the field reaction to stem rust, as determined by percentage infection, in these crosses is mainly if not entirely controlled by the factors which govern the inheritance of the seedling reaction to form 21 in the greenhouse, as determined by pustule type."

Similar investigations have been made with *Puccinia triticina*. Johnston and Melchers (20) studied the relation of leaf rust infection to age of wheat

plants and found that a number of varieties that were susceptible as seedlings became highly resistant to *P. triticina* form 9 at heading time. The greatest resistance appeared in the uppermost leaves, so that the infected flag leaf often was flecked while the lower leaves were covered with numerous uredia.

Scheibe (28) reported that the seedling resistance to *P. triticina* characterized by a type 1 or type 2 infection changed to an increased resistance as the individuals grew older and that the higher-inserted leaves were more resistant than the lower-inserted ones. But the varieties with extreme susceptibility and a type 4 reaction and those with extremely high resistance and a type 0 reaction retained their characteristic reactions throughout all growth stages. The increase in resistance in the varieties with low resistance in the seedling stage seems associated with the photosynthetic processes. A decline in photosynthetic activity seems to induce a more rapid development of the telial stage of the rust.

The equatorial countries offer opportunities for further observations on stem-rust infection. Although certain varieties of wheat are resistant to stem rust in the field and even in the greenhouse during their later developmental stages in Canada and the United States, these same varieties are completely susceptible when grown in Peru, South America, and Kenya Colony, East Africa. Abbott (1, 2) has reported that wheat varieties, resistant to stem rust in the fields of northern United States and Canada, are severely rusted in the experimental fields at Lima, Peru. Abbott's plantings were made in June and August, during the winter season when there are heavy fogs and mists on the Peruvian coast. One hundred per cent of Khapli emmer and Hope wheat plants were rusted. Pustules appeared on all plant parts and, although relatively small, often were so numerous on Khapli culms as to coalesce and completely girdle the stem. Several possible explanations have been offered for such differences in rust infection of older plants. Abbott himself (1, 2) suggested that a new and virulent physiologic form of *P. graminis tritici* occurred in Peru, a form similar to form 15, except for its severe attack on Khapli emmer. Some workers in the United States have intimated that there may be strains of host varieties as well as strains of the parasite. However, the seed of Hope used by Abbott was sent to South America from Minnesota, and seed of the same lot produced plants that resisted rust in the United States. It also was suggested that under the environmental conditions described by Abbott the stomatal movements of the varieties in question might permit the fungus to enter the host tissues easily, whereas in the United States the environment often affected stomatal movements of these varieties to such an extent as to exclude the parasite. Altitude also has been considered a factor affecting the rust infection of wheats grown in the equatorial regions, for Abbott (3) noticed

a gradual decrease in stem-rust infection at elevations greater than 8,000 or 9,000 feet.

G. J. L. Burton of Kenya County, East Africa, in correspondence with E. C. Stakman, expressed the belief that altitude probably is a less important factor in rust resistance or susceptibility than people are inclined to believe, its principal rôle being to modify climate sufficiently to enable wheat to grow at the equator. Burton also suggested that photosynthesis, length of day, and intensity of sunlight may be associated in some way with the "failure of certain wheats to show mature plant resistance to stem rust under equatorial conditions" in Kenya Colony. During the "long rains" season from March through May the rusts are severe, but there is little rust during the "short rains" season of November and December.

Gassner (8), on the basis of his experimental work in Uruguay, reached conclusions entirely different from those of most of the preceding workers. His experiments were made in the field, but were so planned and the plantings so arranged as to eliminate as far as possible inequalities of external factors, particularly climate. There was a marked increase in susceptibility to *Puccinia graminis* and *P. coronifera* as the age of the wheat and oats plants increased, the plants that had not reached the shooting stages often having less infection than the older plants, so that there appeared to be a period of "youth resistance" rather than a mature plant resistance. With respect to *P. triticina*, on the contrary, the wheat varieties were markedly susceptible in their early growth stages, but developed a more or less decided resistance as they reached the shooting and flowering stages. Older plants were less resistant than those in the intermediate stages. Gassner and Kirchhoff (9) repeated and amplified Gassner's South American experiments to determine the applicability of the earlier results to conditions in Germany. Although the climate in Germany did not favor the continual seeding experiments, their results were in accord with those secured in South America.

As concerns experience in the United States, there seems little evidence that seedlings are really more resistant to *Puccinia graminis* than older plants. The senior writer's own observations and those of several other observers indicate that seedling plants often become heavily infected when there is sufficient inoculum. The senior writer, for example, has repeatedly seen wheat seedlings very heavily infected in her own field plots. The uredia were entirely normal in appearance, without any indication whatever that the plants had "juvenile resistance." Furthermore, large, erumpent, very healthy uredia often appear on seedlings of wheat in the fall in the Southern States and in the early summer in the Northern States. In the fall of 1934, rust was fairly abundant on volunteer seedlings of oats and wheat in Minnesota. It seems likely that seedlings often escape infection because of relative paucity of inoculum at the time they are growing. Long

experience in culturing stem rust in the greenhouse at St. Paul, Minnesota, certainly does not lend support to the idea of "juvenile resistance." On the other hand, there is abundant evidence that some varieties of wheat tend to become more resistant with age. Varieties resistant to stem rust in the seedling stage in the greenhouse are resistant in the field also, although the converse is not always true. These facts lead the writers to the conclusion that seedlings of the small grains are as susceptible as older plants, or, in the case of some varieties, even more so.

Hope wheat has been resistant to stem rust in the field throughout the Mississippi Valley of North America since 1926, when it was first selected from the Marquis × Yaroslav emmer cross. It seldom had more than a trace of stem rust until 1933, although it had been grown under conditions of artificial stem-rust epidemics for several years. Its field resistance was so marked that plant breeders, using Hope or H–44–24 as rust-resistant parents, have relied upon the field reactions and the "mature plant resistance" in breeding work (6, 13, 14, 15, 16). Clark and Humphrey (6) considered the near-immune reaction of Hope as a new character inherently distinct from resistance or susceptibility and found it to be inherited as a dominant character.

The necessity of determining the nature of resistance to stem rust in Hope wheat is apparent, especially if Hope is to be used to a considerable extent in breeding for rust resistance. If the maturing plants of Hope continue to have a marked field resistance to stem rust over a period of years in the United States it must be determined what factors bring about the change from the complete susceptibility of seedlings to some of the rust forms to the great resistance to all rust forms in the older plants. It also is necessary to explain more fully the rust resistance of Hope in the northern hemisphere and its susceptibility in the southern hemisphere. Is it likely that in the future Hope may be susceptible to rust in the United States?

The present study was made for the purpose of determining as definitely as possible the basis for the marked rust resistance in the field of maturing plants of Hope wheat.

FIELD OBSERVATIONS

In 1933 there was more stem rust on Hope wheat grown in the rust nursery plots at University Farm, St. Paul, Minnesota, than ever had been observed before. Some of the plantings had as much as 35 per cent stem rust, the infection types being recorded as varying from R (resistant) to S (susceptible). In every case the large normal pustules, recorded as S, were on the lowest internodes or just above the nodal swellings of the central internodes, the tissues with the greatest free water content and the most shaded and protected parts of the plants in the field. A little higher on the culm,

or on the basal parts of the central internodes were pustules with heavy spore masses but with the surrounding host tissues blackened or purpled with anthocyanin. Still higher on the leaf sheath the pustules were smaller and the pigmentation greater, and often the leaf-sheath portion an inch or two below the blade was covered with subepidermal pustules or mycelial infections, while those host tissues were soon dried or scalded. On the peduncle and awns the pustules varied from minute but normal pustules on the greenest and most succulent parts to hypersensitive flecks, with the latter type of infection predominating. Short young volunteer culms that were green and vigorous when the rest of the plants were ripenings were completely susceptible to rust, with very little chlorosis or pigmentation.

During the growing season of 1933 at St. Paul there was an abundance of sunlight and very few cloudy days. Temperatures were extremely high during the last half of June and moderately high during July. All wheats were exposed to drying winds, which, with the heat, hastened ripening. Despite the weather there was a heavy epidemic of stem rust and ample opportunity to study evidences of resistance in the field.

Other varieties in the plots were examined to determine whether there was similar rust infection in a number of wheats or if the phenomenon was peculiar to Hope. There was no evidence of hypersensitiveness, pigmentation, or discoloration around pustules on Marquis, Haynes Bluestem, Reliance, Reward, Ceres, Kota, Supreme, Progress, Marquillo, Webster, Mindum, or Nodak. There were some evidences of discoloration, pigmentation, and subepidermal pustules on Pentad and on Kubanka. On Acme, H–44–24, H–35, and on Khapli and Vernal emmers, the reactions were similar to those on Hope. There were all variations of infection, and certain of the host tissues seemed more sensitive to drying and scalding, as was the case in Hope. When various hybrids were examined without immediate knowledge of their parentage, it was observed that some reacted like Hope, while others were without any signs of discoloration or pigmentation. In every case of a hybrid with reaction similar to that of Hope it was found that Hope, H–44, H–42, or H–35 had entered into the cross. Not all crosses examined containing one of these four varieties reacted thus, though most of them did.

On culms of *Hordeum jubatum*, collected July 13, 1933,[4] at Ben Lake, Pope County, Minnesota, there were indications of similar reaction to stem rust. Anthocyanins were distributed through most of the culm tissues, surrounding the normal rust pustules on the lower parts of the leaf sheaths and spreading about the hypersensitive and necrotic spots on the peduncles and the upper leaf sheaths. Apparently in Minnesota in 1933 such field reactions to stem rust were not confined to Hope wheat.

[4] Collected by E. C. Stakman, J. J. Christensen, L. W. Melander, and L. M. Stahler.

The reactions of Hope wheat in all stages of development, from seedlings to maturing plants, to physiologic form 21 of *Puccinia graminis tritici* were studied in the greenhouse and field to determine if the field resistance of Hope might be an escape from rust infection, the result of the interplay of environmental factors, or a true resistance.

THE RUST SUSCEPTIBILITY OF YOUNG HOPE PLANTS

Under ordinary greenhouse conditions *P. graminis tritici* 21 always produced type 4 infection on Hope seedlings; and the plants continued to be completely susceptible (Fig. 1) until they were six or seven weeks

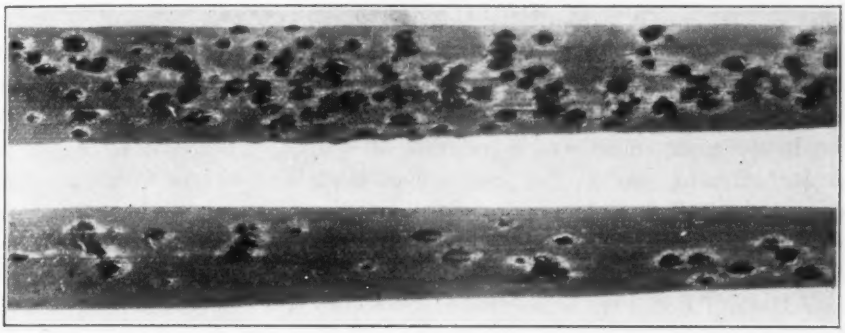

FIG. 1. Leaf blade of Hope wheat 6 weeks old rusted by *Puccinia graminis tritici* 21. The reaction varied from 4 to 4++ with chlorosis.

old, when they had produced five or six succulent green leaves and often were stooling. Uredia were numerous and the fungus sporulated abundantly. Occasionally, the host tissues surrounding an infection were slightly chlorotic, but in no case was there indication of the pigmentation that occurred on Hope in the field and that so often appeared in other hosts resistant to stem rust.

THE RUST REACTIONS OF HOPE IN THE JOINTING, BOOT, AND HEADING STAGES

Older plants, in the pre-boot, the early-boot, or heading stages, were inoculated repeatedly but with very unsatisfactory results, although particular attention was paid to the technic used. Several different methods of applying inoculum were tried, and every effort was made to provide ideal environment for infection. The inoculum was viable and caused severe infection on wheat seedlings inoculated as checks. Temperature varied from 18° to 22° C., soil moisture was sufficient, and the atmosphere remained saturated throughout the moist-chamber period in the greenhouse or for several hours in the field. In many instances inoculations were timed so that the stomata would be open a few hours after the

inoculum was applied, at the time when the greatest percentage of spores presumably had germinated. Inoculum was brushed or shaken over the moistened plants, applied with a flattened needle, or rubbed on with moistened fingers. In the greenhouse the inoculated plants were so placed as to obtain all the sunlight available, especially when experiments were made during the winter months of December, January, or February, the time during which stormy and cloudy weather with the lowest light intensities and the shortest day lengths of the year prevails at St. Paul. Plants were inoculated in the field at different times during the day and evening, and an attempt was made to furnish a saturated atmosphere for a short time after inoculation by enclosing the plants in cloth cages or by wrapping moist cotton loosely around the inoculated culms.

In spite of all technical precautions the older plants seldom rusted. Often there was no sign of infection, especially if the peduncles or the awns had been inoculated. Occasionally, two weeks or more after inoculation in the greenhouse, single pustules or a group of uredia appeared on the leaf sheaths, just at the juncture of blade and sheath or at the base of the sheath close to the node. Such uredia were normal in appearance but never sporulated so abundantly as did the uredia on young Hope plants. In the field in 1932 not even a trace of rust appeared when the older Hope plants were inoculated by brushing or rubbing the urediospores on moistened plants.

In some cases when inoculations were made in the greenhouse chlorotic areas appeared on the culms, peduncles, and awns, but at the time they did not seem to resemble the flecking caused by the rust when the host it attacks is highly resistant to or immune from rust. They seemed more like scalded tissues than rust-infection centers, although later work and the field observations of the following year (1933) indicated that they were infection centers arrested in their normal development. Sometimes the leaf blades seemed entirely scalded after being inoculated and incubated in glass moist chambers exposed to bright sunlight.

The failure to secure more than a trace of rust infection on older plants was difficult to explain. The complete lack of symptoms on so many of the older plants may have resulted from the inhibition of spore germination or germ-tube growth by some toxic substances present in the older plants but not the younger ones. On the other hand, the production of a few small uredia on the most succulent plant parts seemed to belie the acquisition of a protoplasmic resistance to rust by older plants, or at least to raise doubt as to whether certain tissues remained susceptible to rust while young and succulent but suddenly acquired a rust resistance as they approached maturity. To determine the rust reaction of the very young tissues of the older plants, a suspension of rust inoculum was

forced into the leaf rolls and boots of older plants by means of a hypodermic syringe. In addition, experiments were made on the effect of plant extracts on spore germination.

EFFECT OF PLANT EXTRACTS ON GERMINATION OF UREDIOSPORES

The possible presence of a toxic substance in the sap of older plants was investigated by extracting the sap from culms of older plants of Hope wheat growing in the field. The extract was filtered through a Berkfeld filter, prepared in three different dilutions, and the effect of the filtrate on urediospore germination of *P. graminis tritici* 21 studied and compared with the effect of a similar filtrate obtained from culms of the rust-susceptible variety Marquis. The percentage of spores that germinated in the filtrates of different concentration was not so high as for spores in distilled water. However, urediospores germinated as well in extracts of Hope wheat as in those from Marquis, regardless of concentration of filtrate; hence there seemed to be no inhibiting substance in Hope that would prevent the germination of rust spores.

Furthermore, some of the material inoculated by means of the hypoder-

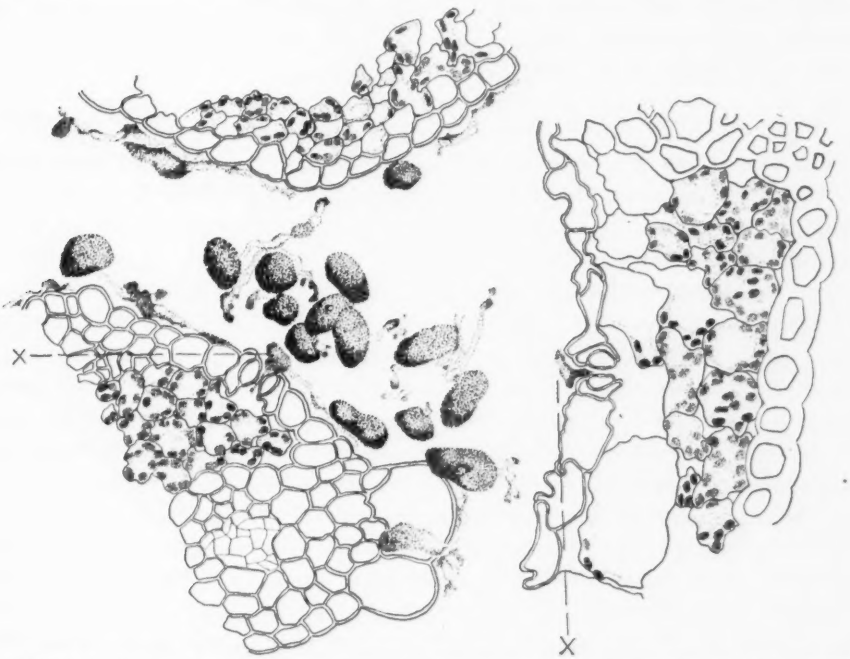

FIG. 2. Cross section of a portion of the boot of Hope wheat 20 hours after inoculation with *Puccinia graminis tritici* 21 by hypodermic syringe. Most of the spores have germinated, and some of the germ tubes have formed appressoria (x) to enter the stomata of the young tissues.

mic syringe was studied histologically and the progress of the rust followed. Spore germination was not inhibited: twenty hours after injection of the spore suspension into the boot of Hope most of the urediospores had germinated and the germ tubes had grown many millimeters (Fig. 2). Some of the tubes were twisted masses in the spaces between sheath and developing head, others had grown along the surfaces of the plant hairs, the awns, or the inner part of the leaf sheaths. There were numerous appressoria over the stomata of the surfaces within the boot, and there seemed no obstacle to entrance of the fungus. The boot provided an ideal moist chamber for spore germination, survival of germ tubes, and the beginning of mycelial infection in the host. Apparently there was nothing to inhibit germination and growth of the inoculum in the Hope host.

A number of investigators (34, 38, 39) found that intense sunlight inhibited spore germination, but it is not likely that this accounts for the lack of infection in older plants of Hope, because seedlings subjected to the same inoculum and the same moist-chamber conditions always were severely infected. To determine whether the inoculum was effective, the epidermis was stripped from the leaf blades of older plants of Hope at various intervals after inoculation. In all cases a relatively large percentage of spores had germinated and formed appressoria over the stomata, so it cannot be said that germination was inhibited.

EFFECT OF INOCULATING OLDER PLANTS BY MEANS OF HYPODERMIC SYRINGE

A number of inoculations by means of hypodermic syringe were made in the greenhouse and in the field to test further the acquisition of rust resistance or the existence of a sharp turning-point with respect to rust reaction as the wheat tissues matured. When the hypodermic syringe was used to force a suspension of urediospores into the rolled leaf sheaths or the boots of Hope plants growing in the greenhouse, the resulting prevalence of infection was usually moderate or severe, and appeared to depend somewhat on the age and succulence of the tissues subjected to attack. The heads inoculated when newly formed and several days before emergence from the boot were completely susceptible and rusted severely in all chlorophyllous tissues. If inoculated just a day before emergence from the protecting leaf sheaths, the infection was light to moderate; some of the uredia were normal in appearance but small, and there were numerous chlorotic areas and evidences of mycelial infections without formation of uredia. Infection of the leaf sheaths, too, seemed severe if the inoculation was made early enough to permit the rust inoculum to reach the young tissues several days before they unrolled. When infection occurred on the expanded leaf sheaths, particularly on the outer layers, it varied from light to moderate, with considerable anthocyanin pigmentation and a browning or blackening of tissues around

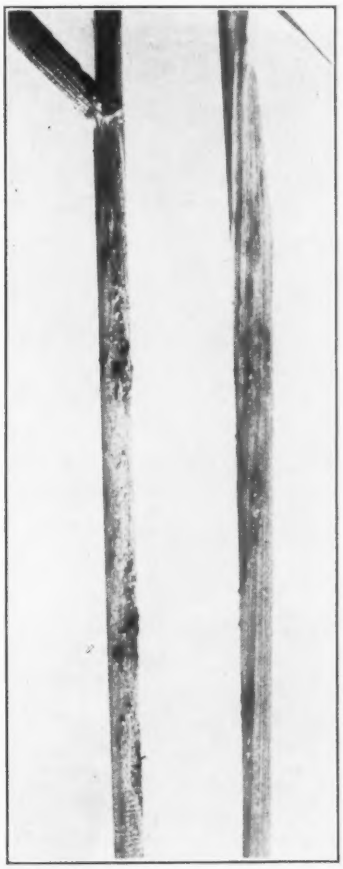

FIG. 3. Maturing plants of Hope wheat rusted by *Puccinia graminis tritici* 21 after inoculation with the hypodermic syringe. Pustules are small, sporulation is scanty, and there is considerable pigmentation about the infection centers when the plants are exposed to intense sunlight.

the pustules (Fig. 3). Dark streaks sometimes appeared on the inoculated leaf sheaths, and one was uncertain, before microscopic examination, whether they were rust infections. Most of them were mycelial infections or subepidermal uredia containing a few spores and a considerable amount of mycelium (Fig. 4, A). A number of the host cells had been killed by the parasite, and the tissues of the infected areas often were filled with materials resembling phenolic deposits. These infections usually remained subepidermal, but sometimes the epidermis was wholly or partly broken and spore production increased slightly. The outward appearance of a pustule was more or less normal, but spore production still was very limited and the

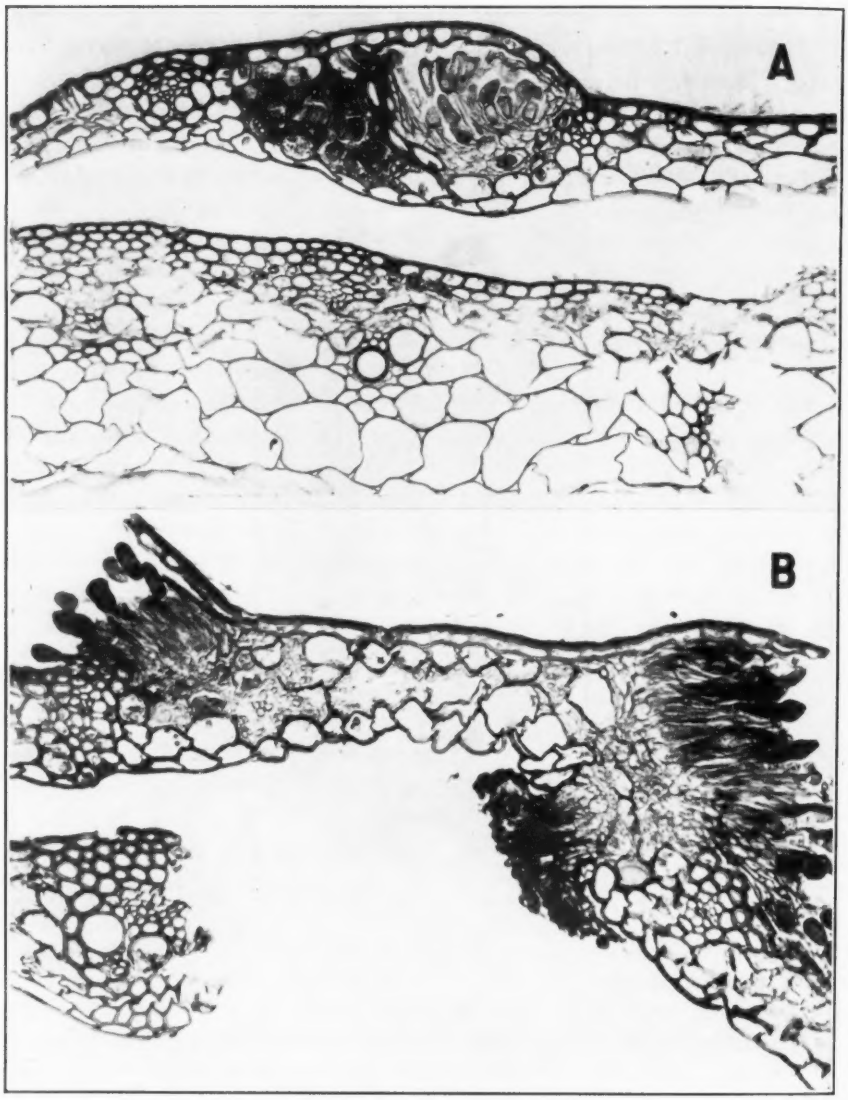

FIG. 4. A. Cross section of leaf sheath of Hope wheat rusted by *Puccinia graminis tritici* 21 after inoculation by hypodermic syringe and exposure to intense sunlight. Subepidermal pustules with abundant mycelium but relatively few spores. Many of the host cells have been killed, and pigments have been deposited in part of the infected tissue. B. Inner rolls of culm of Hope wheat rusted by *P. graminis tritici* 21 2 weeks after introduction of inoculum by hypodermic syringe. Pustules are normal, with abundant mycelium and sporulation, and with very little pigmentation and no destruction of host cells.

surrounding host tissues were brown, black, or deep purple. Only a few pustules ever produced spores in abundance.

Apparently, Hope was definitely antagonistic to rust and repressed its development. It seemed as though Hope acquired resistance to stem rust as its tissues aged, the youngest and most succulent parts of all older plants remaining completely susceptible to rust, while tissues just a few days older resisted infection.

Similar results were secured as a result of hypodermic-syringe inoculations in the field in 1932, but the variation in infection was more pronounced. Occasionally necrotic flecks were the only signs of infection. Eleven days after inoculation, when many uredia normally would be rupturing the epidermis, there often were only chlorotic areas and hypersensitive flecks on the awns and glumes of heads that had emerged from the inoculated boots and a few dark spots on the inoculated leaf sheaths. Infection sometimes occurred on the culm, but the uredia always were extremely small and the surrounding tissues deeply pigmented. Normal, well-developed uredia, however, did not appear on the outer rolls of the culms inoculated in the field during 1932.

Nevertheless, when the inner rolls of the culm were examined a number of normal rust pustules were present (Fig. 4, B), hidden beneath and protected by the outer rolls of the sheath. In such sections there were abundant mycelium and numerous spores, the host cells were not killed, and there was less pigmentation. Host tissue of the same age as that of the outer sheath rolls, in fact the enrolled part of the outer sheath protected by its overlapping margin, permitted normal development of the rust fungus, while the exposed part of the same sheath resisted attack. In the protected inner rolls development was not repressed in any way, possibly because some pathogen had access to the outer rolls and inhibited rust there but was unable to attain the inner rolls, or because some environmental factor affected one tissue more than the other.

EFFECT OF OTHER ORGANISMS ON DEVELOPMENT OF STEM-RUST INFECTION IN HOPE WHEAT

Some of the discolored tissues resembled lesions caused by *Bacterium transluscens* var. *undulosum*, and a few of the blackened pustules looked as though they were parasitized by the bacterium noted by Levine, Granovsky, and Leach (22). Isolations were made from the affected parts to determine whether some other organism was responsible for repression of the rust. In a very small number of the suspected cases the bacterial parasite of rust was isolated, but it was not sufficiently wide-spread to account for the type of rust infection that occurred on Hope wheat. Blackchaff bacteria were never isolated from the rusted culms. Nevertheless, the effect of blackchaff on rust

development was determined by introducing simultaneously a suspension of bacteria from a pure culture of *Bacterium transluscens* var. *undulosum* and a suspension of rust urediospores into young and succulent shoots of Hope wheat plants varying in age from 4 to 10 weeks. Blackchaff symptoms appeared in three days and rust pustules formed in immediately adjacent tissues in six days. Apparently, neither pathogen deterred the other from parasitizing wheat tissue.

A Helminthosporium was isolated from some of the discolored tissues, but it appeared to be a secondary invader. A suspension of its spores, together with a suspension of rust urediospores, was forced into growing wheat plants. Helminthosporium lesions developed not far from the needle punctures, but, as in the case of blackchaff, the development of rust was not inhibited. Apparently, the seeming resistance to rust in older plants of Hope was not due to the antagonism of any other parasitic organism.

Attention then turned to environment and its possible effect on Hope and rust infection. It seemed certain that environment favored spore germination and entry of the parasite, but there was the possibility that establishment and development of the parasite was affected by environment, or that the host was more easily affected by environment and in turn repressed the rust. The outer rolls of the inoculated culms seemed particularly sensitive to light and temperature, for, as was mentioned previously, the leaf blade and part of the sheath often were dry and scalded after a few hot days in the field or after having been in a glass moist chamber in the greenhouse during hot bright days.

It seemed highly probable that some such environmental factor as intense light or high temperature, might decrease the succulence of the exposed plant parts more rapidly and disastrously than it affected the protected tissues, or that the succulence of Hope tissues was more easily affected by environmental factors than were tissues of the other wheats. The fungus could not be expected to develop normally without available water and nutrients. But, when corresponding sections of Marquis and Hope wheats, growing in the field under different conditions of light and soil moisture, were compared as to their free water content, the differences between the two varieties were too slight to account for the repression of rust in Hope and the normal and luxuriant development in Marquis. Hope, when grown at University Farm and not attacked by rust, was not markedly more sensitive to its environment than was Marquis.

There also was the possibility that Hope wheat carried a factor for pigment production under certain environmental conditions and that the pigments in turn prevented the normal establishment of the rust parasite. Broadfoot and Robertson (5) noted the production of pigment in Reward and Marquis wheats, some strains of which seem to carry factors for pigment

production under intense light. In the present work, however, the authors did not observe any pigmentation in nonrusted plants of Hope grown under intense sunlight. Only the plants that had been inoculated with rust produced pigments in the tissues surrounding the center of attack.

Environmental factors influence the parasite directly, but from the observations at hand, it is unlikely that there was an inhibition of spore germination that might explain the lack of infection in so many experiments. Light may repress the mycelial development or may stimulate sporulation before the fungus has had opportunity for ample vegetative growth. But, if such were the case, the phenomenon probably would occur with certain rust forms regardless of the host they were attacking.

Another possibility is that environment affects the combination of host and fungus more easily than it affects either one alone. As soon as parasitic relations are established the two component parts are merged into an entirely new entity. The interplay of environment and the new entity may be quite different from the interplay of environment and fungus or environment and wheat plant. In the present work the two environmental factors, light intensity and temperature, were studied in relation to the development of rust infection in Hope.

DEVELOPMENT OF RUST INFECTION UNDER REDUCED LIGHT INTENSITY

The first experiments under reduced light intensity were made during the last days of March with Hope wheat plants thirteen weeks old, with numerous tillers and with the last leaves shooting, but with no indication of the swollen boot. The day chosen for inoculation was one of alternating cloudiness and diffuse sunlight. The moist chambers were of glass, but they were shaded by screens of a single layer of cheesecloth and not exposed to direct sunlight. The inoculum was rubbed over all plant surfaces with moistened fingers, and care was taken to keep the surfaces moist and the atmosphere saturated, and to keep the temperature as uniform as possible. After 48 hours in the moist chamber the plants were removed but remained screened from direct light. Fortunately, perhaps, there were 16 successive cloudy days following inoculation. After that time the rust infection was well distributed over the entire plant; uredia were scattered from tip to base of leaf blades and over all parts of both lower and upper internodes. Uredia on the more shaded lower internodes were of type 4, while those on the upper parts were generally of type 3 with some chlorosis about each pustule, and occasional uredia were smaller than type 3. The Hope and Marquis seedlings inoculated as checks were severely rusted, with type 4+ and 4++ infection respectively. By the twenty-fifth day all pustules on the older Hope plants were of type 3+ or 4. The period from the twenty-second to the thirtieth day after inoculation was one of bright sunshine and during

that time spore production in the well-established rust pustules was tremendously increased, so that infection was as severe as that that usually developed on protected young and succulent tissues inoculated by hypodermic syringe and approached the severity of infection generally obtained in seedlings.

Later, similar experiments were made, with the light intensity reduced and diffused by cheesecloth screens. Plants in the pre-boot, in the boot, and post-head stages were inoculated, sometimes by brushing with rusted seedlings but more often by rubbing on the inoculum with moistened fingers.

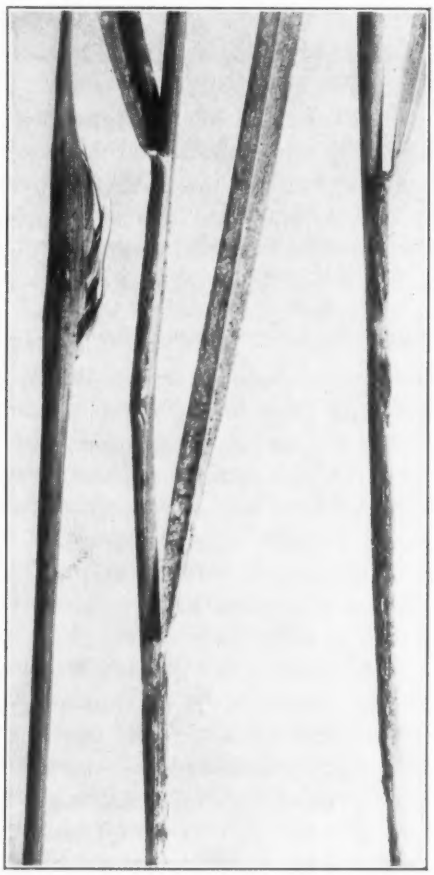

FIG. 5. Culms of Hope wheat rusted by *Puccinia graminis tritici* 21 after inoculum had been rubbed on the plants and the plants shaded from intense sunlight. The two culms at the left were photographed two weeks after inoculation before the fungus reached the height of its spore production. The culm at the right was photographed 4 weeks after inoculation, and, although the ruptured pustules were not large, spore production was so great that the entire culm was covered with the spore mass.

Check plants, either seedlings or plants 3 to 4 weeks old, always were inoculated and incubated with the older plants. In some experiments spore germination was tested as well. Inoculations were made on cloudy days whenever possible, and in a number of cases the stomata were examined during the first few hours of the moist-chamber period. Rust pustules first appeared 8 to 9 days after inoculation on the shaded plants, but the infection did not attain its greatest severity until 3 to 4 weeks after inoculation (Fig. 5). All plant parts that were green and succulent at the time of inoculation proved to be susceptible to rust attack. The largest pustules were on the more shaded parts and those that seemed to retain their succulence longest: the extreme base of the leaf blade, at the juncture of the blade and leaf sheath, and the leaf-sheath tissue just above the nodal swellings throughout the lower half of the culm. The effect of light intensity was particularly striking when infection on shaded plants was contrasted with the infection on similarly inoculated plants exposed to full and direct sunlight a few days after removal from the moist chamber. The necrosis and the hypersensitive areas and the definite pigmentation on the latter were noticeable about 10 days after inoculation. On the shaded plants rust infection developed slowly but certainly, and after 3 to 4 weeks urediospore production was so great that the entire culm seemed covered with the spore mass.

When rust inoculum was forced into the expanding shoots by means of a hypodermic syringe, all parts of the shaded older plants were completely susceptible to rust, and severe infection developed within two weeks after inoculation (Fig. 6). Type 4 pustules predominated, and there was no indication of resistance to rust. The outer culm tissues, as well as the protected inner sheath rolls, were severely rusted, and there was no evidence of pigmentation or hypersensitiveness. Hope seems to have no protoplasmic resistance to *Puccinia graminis tritici* 21 when light intensity is reduced.

There are indications that other fungi may have optimum light intensities for development on their respective hosts. Volk (35) found that a slight reduction in light intensity allowed a more luxuriant growth of *Erysiphe graminis* on rye but hindered the infection of rye by *Puccinia dispersa*. He also obtained better development of the facultative parasite *Cladosporium fulvum* on tomatoes when the light intensity was reduced slightly.

The development of rust infection usually is slower in older plants than in seedlings, partly because of the differences in cell-wall structure of the rust-susceptible parts, partly because of the differences in the plant colloids and their effect on the water relations and nutrition of the parasite. It is possible that environmental factors, such as light intensity, may affect the normal rate of change in host constitution sufficiently to permit luxuriant

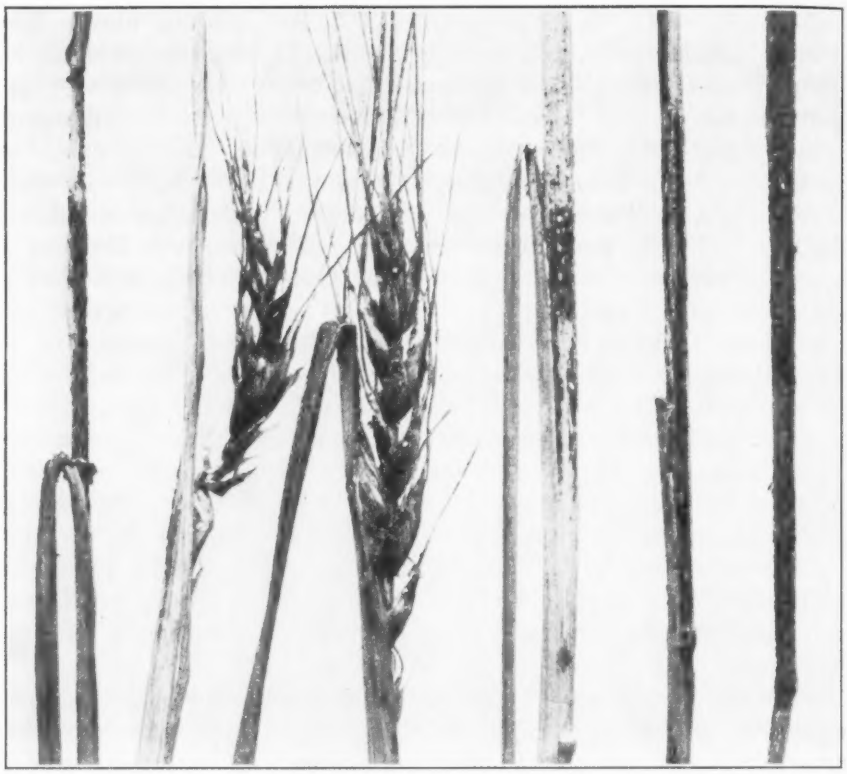

FIG. 6. Hope wheat plants rusted by *Puccinia graminis tritici* 21 after inoculation by hypodermic syringe. Plants were shaded from intense sunlight after inoculation and all parts proved to be completely susceptible to rust. Most of the pustules were large, well-developed, and sporulated abundantly.

development of the pathogen at one time or to impede its development at another. Temperature, likewise, may affect the compatibility of parasite and host, particularly in certain delicately balanced parasitisms.

EFFECT OF HIGH TEMPERATURE ON RUST INFECTION

Older plants of Hope were inoculated with *Puccinia graminis tritici* 21 by rubbing urediospores over the moistened plant surfaces. Plants were in the pre-boot, the early boot, very late boot, or had headed just a day or two before inoculation. Inoculated plants were placed in moist chambers, shaded from direct light, and kept at 24° C., to permit spore germination and entrance of germ tubes. After 48 hours the plants were placed on the greenhouse bench and again shaded. The crocks were buried in moist sand so that the soil might be slightly cooler than the atmosphere, but the daily air temperatures in the greenhouse at this time usually varied from 26° to

33° C., and the relative humidity of the air seldom was more than 20 per cent. Light rust infection appeared thirteen days after inoculation. At that time there were chlorotic and necrotic areas on the awns, glumes, and peduncles that had been inoculated and on a few of the leaf blades and sheaths. Small pustules appeared scattered over the lower internodes, on the upper internodes just above the nodal swellings, on some of the leaf blades, and on one glume. Fifteen days after inoculation it was evident that the plant tissues were drying as a result of the high temperature, despite the shade and an abundant supply of soil moisture. Rust infection was light, and the severity did not increase during four weeks after inoculation. At that time many of the host tissues were thoroughly dry and scalded and rust was evident only on those parts that had retained their chlorophyll and some degree of succulence. There were a few groups of pustules of type 3 = on the north side of two of the greenest culms, but most of the pustules were minute and there was an appreciable amount of pigmentation and chlorosis. A number of infected areas were blackened but spores never appeared, and hypersensitive flecks were common. The infection resembled that under intense light.

This is further evidence that environmental factors influence the complex and dynamic phenomenon of parasitism and that each combination of host and parasite must be considered individually, as an optimum environment for one combination may be detrimental to another. In this respect Hope wheat and *Puccinia graminis tritici* 21 seem to be more easily affected by environment than some of the other combinations studied. Furthermore, Smith (29) has shown that, in Washington, the combination of Hope wheat and *Tilletia tritici* is greatly affected by environment, especially by temperature. Spring-sown Hope is resistant to bunt; when fall-sown it is susceptible. Smith found that temperature is likely to rise very soon after emergence of wheat seedlings in the spring, but in the fall the rise in temperature is delayed for several months. In his experiments high temperatures checked the development of bunt in Hope wheat but not in the variety Jenkins. Smith thinks that the internal composition or the protoplasmic organization of Hope at low temperatures permit the fungus to develop, but that the different composition at high temperatures inhibits or retards the fungus.

DISCUSSION

The foregoing observations support and strengthen the opinion that a host-parasite complex is affected quite as much by environmental factors as are its component parts. It is probable that the balance between host and parasite may be affected more easily than has been realized in the past. The micro-environment, particularly, may determine the develop-

ment of an individual rust infection and the host's reaction to its parasite. In the past it often has been the practice to study the direct effect of environmental factors on the parasite or on some phase of the infection, and to consider the host in its protoplasmic constitution, its vigor, or its physiological development, merely as another factor influencing the progress of the parasite. Recently, however, Johnson (19) has called attention again to the necessity of considering the host-parasite complex when studying cereal rusts, recognizing the fact that environmental factors may affect the interactions of host and fungus.

The combination of Hope wheat and *P. graminis tritici* 21, as studied in the present work, seems to be easily affected by its environment. There was the possibility that the metabolism of Hope, a variety produced by crossing an emmer and a common wheat, was more affected by environment than were metabolic processes of other varieties, and that the resulting changes in the host in turn affected the development of the fungus. This view is supported in part by the work of Smith (29), but in the present work, Hope, without its stem-rust parasite, is less sensitive to its environment than is the host-parasite complex.

Non-rusted Hope plants under intense sunlight did not form the pigments that so generally characterized the tissues surrounding rust infections in Hope. The presence of the parasite was essential for the coloration, intense light alone being insufficient stimulus for the reaction. Nor was the parasitic presence alone responsible for the coloration, because, with reduced light intensity, the pigments were not produced.

Hope seems less resistant to drought than do some of the other wheats; and it was thought at first that the rapid drying and decrease in succulence of the leaf sheaths might be peculiar to Hope, even when there was no opportunity for rust infection. This was not true, however, for when free moisture contents of various tissues were compared in the varieties Marquis and Hope, the determinations ran approximately parallel throughout the important part of the growing season, from the time the plants were in the jointing stages until after flowering. Here, too, both the presence of the fungus and the intense sunlight were required to bring about the rapid drying of tissues.

The interactions of rust and wheat cells are not at all clear and at present it is impossible to explain the effect of external factors on the complex. It may be that the attack of the parasite is so violent and rapid as to destroy the photosynthetic apparatus of the host cells and make it impossible for them to assimilate materials for their own tissues and the fungus as well. It may be that the rust upsets the water relations or nutrient conditions of the wheat tissues. If the fungus appropriates water or nutrients faster than the supply required by the host can be replenished.

it would not be surprising if the host tissues dried out before the fungus was able to complete its development and sporulate. There is the possibility also that the establishment of the parasite or any disturbance by it of water or nutrient relations of the host plant might also affect the rate at which the colloidal materials of the wheat change; and, after the initial disturbance, external environmental factors in turn may have a greater effect on the colloidal condition and processes of change in the protoplasm. Undoubtedly, changes in metabolic rates or disturbances of the physiological processes in host and parasite influence the compatibility of the two organisms. In the light of recent work it is impossible to say whether the fungus entity or the host entity is the more important. Both are requisite and, in the opinion of the writers, probably should be considered as a complex in the future studies on the nature of rust resistance.

Other workers also have noted changes in resistance to rusts as the environmental factors of light and temperature varied.

From epidemiology studies of *Puccinia graminis* and physiologic-form surveys made annually in the Mississippi Valley, it is evident that environmental conditions affect rust infection, sometimes to such an extent that it is difficult to distinguish between certain physiologic forms. Stakman, Levine, and Wallace (32) point out that forms 11 and 32 of *P. graminis tritici* might easily be confused under certain environmental conditions. Form 11 produces a type 4 infection on the durum differentials, which may appear like the type X produced by form 32 unless environment is very favorable for rust development. The same difficulty is encounterd in differentiating forms 38 and 39 (see Stakman and Levine, 31), for the type X infection produced on the durum differentials by form 38, under certain environmental conditions, is not easily distinguished from the type 4 infection produced by form 39. Wallace (36) also has discussed the similarity of forms 18 and 36, which are identical in their behavior on all differential varieties with the exception of Kubanka. Form 18 produces type 4 infection on Kubanka and form 36 the type X infection, but, under certain environmental conditions, the flecks and small pustules of the type X infection seem to be masked by the large pustules, and form 36 produces what appears to be type 4 infection on Kubanka. Wallace had difficulty distinguishing between forms 18 and 36 in the physiologic-form surveys of 1926 and 1928.

Johnson (19) also believes that many of the wheat varieties now commonly grown may vary in their rust resistance in accordance with weather conditions. He decided that temperature, and probably light, influenced profoundly the X reaction produced on both seedlings and older plants of durum wheat varieties with a number of physiologic forms of *P. graminis tritici*. Under low light intensities the typical X reaction was produced at the intermediate temperature of 65° F., but at lower temperatures (55 to

60° F.) the reaction was commonly O; or X, while at higher temperatures (70 to 75° F.) the reaction usually was 4. An increase in light intensity permitted the development of the typical X reaction at the lower temperatures, while the increase in temperature brought about the development of the 4 reaction. Johnson's results agree with those of Waterhouse (37) in Australia, who found that seedling reactions with certain physiologic forms of *P. graminis tritici* varied from type O; in winter to a type X in spring and to a type 4 in summer.

Gordon (12) reports that Joanette strain of oats varies greatly in its reaction to certain forms of *Puccinia graminis avenae,* being extremely resistant to form 4 at 57.4° F., and completely susceptible to the same form at 75.4° F.; and Peturson (26) obtained similar results with some physiologic forms of *P. coronata avenae,* three oat varieties being completely susceptible to form 7 at 77° F., very resistant to the same form at 70° F., and practically immune from this form at 57° F.

The work done by Melander (23) with *P. graminis tritici* indicated that at the very low temperature of 0 to 1° C., physiologic form 35 on Little Club wheat and form 15 on some of the wheat differentials produced pustules approaching a type 1 infection. This type prevailed as long as the wheat seedlings were kept at the low temperature, but if they were transferred to 20° C. the pustules changed to type 3 or type 4 uredia.

Thus in some rusts relatively high temperatures are necessary for susceptibility with the complete development of the parasite, but low temperatures tend to swing the reactions towards resistance. On the other hand, high temperatures may be detrimental to the development of rust infection and thus be responsible for a field resistance to rust. In Germany Gassner and Straib (10) found that development of and the degree of resistance to *P. glumarum tritici* depended greatly on temperature relations: wheat varieties resistant at 20° C. were severely rusted at slightly lower temperatures. Radelescu (27) believes that external factors may affect and change the physiologic resistance of some wheat varieties to *P. glumarum tritici,* and his work indicates that temperature has been one of the primary factors responsible for the field resistance of wheat to stripe rust in Germany. In the western provinces of Canada, too, temperature seems to have a profound effect on the reaction of wheat varieties to stripe rust. Newton, Johnson, and Brown (25) tested a number of varieties and found them all resistant to stripe rust at 78° F., but that many varieties were completely susceptible at 54° F.

Similar observations have been made for leaf rust of wheat. Gassner and Straib (11) found that certain wheat varieties that were highly resistant to *P. triticina* 14 at about 20° C. were completely susceptible to form 14 when cultured at lower temperatures.

In many of the cases cited it is impossible to decide whether the effect of temperature is on the parasite or on the host-parasite complex. The effect of temperature on germination of spores and growth of germ tubes of various rusts is definite and well established; but as soon as the parasitic relationships are set up in the host plant it is impossible to consider the rust as a separate entity any longer.

Miss Forward (7) approached the problem of rust resistance from the standpoint of the host plant rather than the pathogen, attempting to study the physiological basis of compatibility and the modifiability of infection type by altering the metabolism of the host. She has reported upon the modification of infection by *P. graminis tritici* when the host metabolism is altered by extended periods of darkness or darkness broken by short exposures to light. There is a loss of compatibility between fungus and host, and resistant symptoms appear, with an interposition of a more or less extended period of darkness at the beginning of pustule formation. On the other hand, darkness broken by a daily 4-hour exposure to light did not visibly alter the metabolism of the host, and rust developed as rapidly under such conditions as when plants were exposed to light all day.

Puccinia glumarum, too, in its uredial stage is affected by light as well as by temperature, for Bever (4) found that with a day length of 15 hours or more the infection type on Pannier barley changed distinctly from type 4 to type 0. Evidently there is an optimum light intensity for development of stripe rust in the field, for Bever mentions that a day length of less than 12 hours lengthened the incubation period for rust in his experiments; and he believes that the tardy spring advent of stripe rust that sometimes occurs in Idaho during cloudy and rainy seasons is due to the reduction in light intensity below the optimum rather than to variations in temperature.

The present work also supports the view that parasitic relations may be profoundly affected by environment; and it emphasizes the fact that the influence exerted by a particular factor of the environment varies greatly according to the rust species considered, and even according to the different combinations of certain physiologic forms of a single rust species and their host varieties. It seems that every individual host-parasite complex has its optima with respect to various environmental factors—optima that may be nearly identical with the optima for numerous other host-parasite complexes, or may be at variance with those for most combinations.

Much investigational work remains to be done in determining the nature of rust resistance. So far, it has been necessary to observe the phenomenon of parasitism as it occurs and varies in nature or as it has been modified by experimental procedure. The ultimate basis for the compatibility or incompatibility of obligate parasites and their hosts is still to be demonstrated.

SUMMARY

The present work offers evidence that there is an optimum light intensity for mycelial development and sporulation of *Puccinia graminis tritici* in Hope wheat.

Under normal intense sunlight of the Upper Mississippi Valley plants of Hope wheat in various stages of development beyond the seedling stage appear to have considerable resistance to *Puccinia graminis tritici* 21.

When light intensity is reduced by shading the plants they are completely susceptible to *P. graminis tritici* 21, just as they are completely susceptible in their seedling stages.

High temperatures have an effect similar to that of intense sunlight, so that plants appear resistant to rust.

The external environmental factors, light and temperature, seem to influence the host-parasite complex rather than its component parts.

The authors are very grateful to Dr. E. C. Stakman for his counsel and critical advice throughout the investigations and to Dr. J. J. Christensen for his helpful criticism of the manuscript.

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STUDIES ON FRENCHING OF TOBACCO[1]

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(Accepted for publication July 23, 1935)

Among the so-called physiological diseases of tobacco, frenching is one of the most puzzling so far encountered by plant physiologists and pathologists. In Princeton this disease proved to be severe in the winter of 1932 in tobacco grown in greenhouses. During the following summer it occurred extensively in plants in an experimental field. Since that time numerous greenhouse experiments have been carried out in an endeavor, firstly, to prevent the disease, and secondly, to determine factors favoring its development. The object of this paper is to present the results of the experiments and such conclusions as could be drawn from the experimental data.

LITERATURE

Frenching of tobacco is very widespread in occurrence, as its presence has been observed in most of the large tobacco-growing areas of the world. In the United States frenching has been reported in Connecticut (4), Florida,[2] Georgia,[2] Indiana,[3] Kentucky[2] (5), Louisiana (13), Maryland[2] (13), Massachusetts,[2] New York,[4] North Carolina[2] (13), Ohio (13, 18), South Carolina,[2, 5] Virginia[2] (13), West Virginia,[6] and Wisconsin.[2] It has been reported in each of the following foreign countries: Canada,[7] France[2] (17), Italy (22), Russia[2] (17), Dalmatia[2] (17), Rhodesia (9), Transvaal (1), West Africa,[2] and Java[2] (17).

The earliest reference to frenching found in the literature was in a letter by John Clayton (6) to the Royal Society, May 12, 1688. Symptoms of frenching have been described by a number of early workers, including Dodge (5), Killebrew (13), and Allard (2). Clinton (4), in 1914, demon-

[1] Published at the expense of The Rockefeller Institute for Medical Research, Princeton, N. J., out of the order determined by the date of receipt of the manuscript.

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strated that frenching cannot be transmitted to healthy plants by inoculation, thereby showing that it is a noninfectious disease and not a severe form of mosaic. His findings were corroborated by Wolf and Moss (26) in 1919, and by Garner (7) in 1922. Johnson (11) observed that poor soil aeration and low soil fertility were contributing factors favoring the development of the disease.

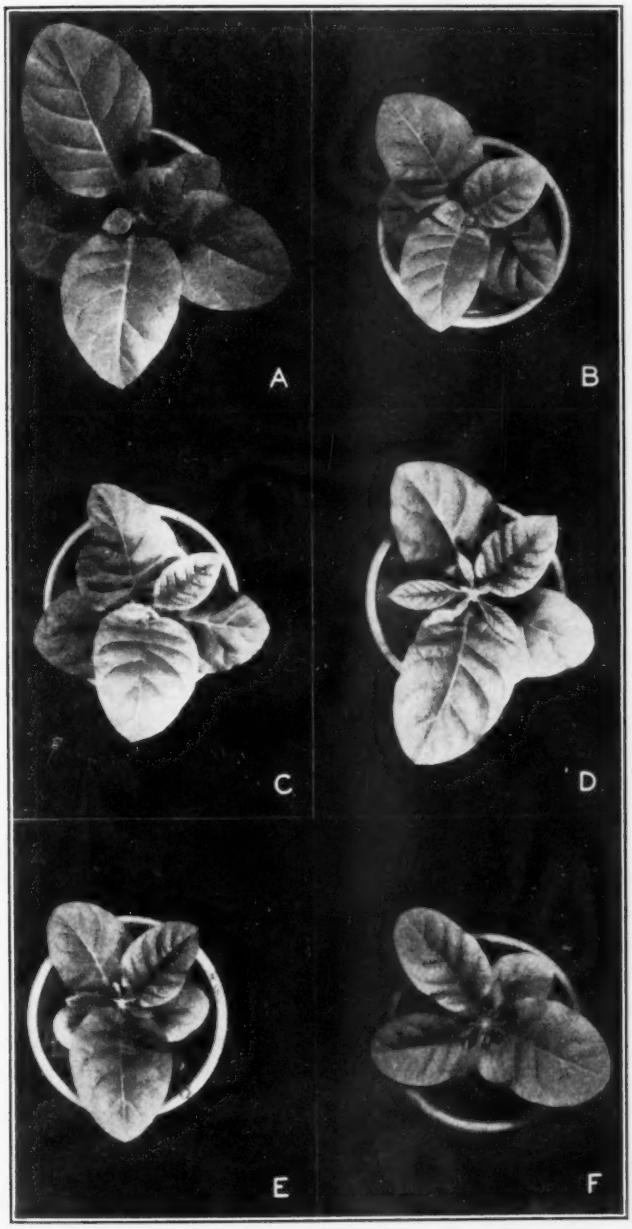
The suggestion that frenching is a nitrogen-deficiency disease was first advanced by Valleau and Johnson (23) in 1926. They concluded that it is the result of an accumulation of carbohydrates brought about by the absence of sufficient available nitrogen. This physiological condition was conceived to give rise to the development of new leaf tissue deficient in chlorophyll. In a later paper they (24) reported that frenching could be readily controlled by the addition of available nitrogen. This conclusion was confirmed by Haas (8) in California and by Hopkins (10) in Southern Rhodesia.

Morgan (16) in 1929 questioned the hypothesis that frenching is a nitrogen-deficiency disease. He was able to show that it developed when various fertilizer mixtures were added to the soil and was not restricted to mixtures deficient in nitrogen. In a later publication Valleau and Johnson (25) modified their original conception that frenching is a nitrogen-deficiency disease by suggesting that it is not caused directly by a deficiency of nitrogen alone, but probably by a lack of balance between translocation of carbohydrates and of nitrogenous compounds within the plant. This condition may be brought about by a deficiency of any nutrient element essential for protein metabolism. In a more recent report Karraker and Bortner (12) suggested that, since frenching is obviously a disease of the growing points of the plant, its expression is the result of a retardation in the translocation of food materials to and from these growing regions.

The possibility that frenching is a toxicity disease rather than a deficiency disease was first expressed by McMurtrey (15) in 1932. He reported in a brief note that symptoms of thallium toxicity in solution culture resembled those characteristic of frenching, although not identical in all respects. Shear (19) also suggested that frenching might be a toxicity disease, but advanced no theory as to the nature of the toxic factor. No conclusive experimental evidence has been presented heretofore to show that frenching is a toxicity disease.

SYMPTOMS

Since frenching has been adequately described by Clinton (4), Shear (19), and Valleau and Johnson (24, 25), only a brief description of the most characteristic symptoms will be given here. The earliest symptom to be observed is chlorosis along the margins of young leaves (Fig. 1, B). This gradually spreads toward the midribs and, finally, the entire interveinal



Photographed by J. A. Carlile

FIG. 1. Tobacco plants showing characteristic symptoms of frenching. A, healthy check plant; B, first symptoms with interveinal chlorosis along margin of leaf; C, interveinal chlorosis extending over entire leaf; D, elongating leaves, forming the strap-leaf stage; E, severe strap-leaf condition; F, stimulation of axillary buds forming a rosette of many, small, strap-shape leaves.

regions are involved (Fig. 1, C). The network of veins, however, remains dark green. As the leaf develops, only the midrib elongates apparently, thereby producing a long narrow, ribbon-like or strap-shape structure (Fig. 1, D). Terminal growth is greatly retarded, resulting in a stunted plant with small, brittle, distorted leaves (Fig. 1, E). In severe cases the axillary buds are stimulated into growth, producing a rosette of small, narrow, strap-shape leaves (Fig. 1, F). These narrow leaves are very light in color. Frenching is not confined to the seedling stage of growth, but may appear at any stage up to flowering. It is sometimes accompanied by a yellowing of the lower leaves.

Symptoms of the disease are noticeable also in the roots. The diameter of the old roots is reduced to about one-half that of normal roots of the same age. The number of root hairs is markedly decreased; in some cases they are completely lacking. The cortex near the root tips is pronouncedly discolored and necrotic. Root systems of frenched plants usually are more profusely branched than those of healthy plants.

EXPERIMENTAL

Numerous greenhouse experiments were carried out in an endeavor not only to control frenching of tobacco but also to determine its causal factor. Since frenching developed very severely in a near-by field, soil from this area was used in all of the experiments except where mention is made of soil from another source. This soil, which will be referred to as field soil, was quite heavy in texture, of high water-holding capacity, and of low fertility, with a neutral or slightly alkaline reaction.

An experiment was first carried out to determine what plants were susceptible to frenching. All seedlings were germinated in sand and then potted in field soil in 4-inch pots. In the first test, seedlings of 16 varieties of *Nicotiana tabacum* L. were used. These varieties, all of which developed frenching within 6 weeks, were Adcock, *angustifolia, auriculata,* Burley, *calycina, chinensis, colossea,* Connecticut Seed Leaf, *gigantea, latissima,* Little Orinoco, *macrophylla,* Maryland, *purpurea,* Turkish, and Zimmers. In the second series 18 different species of the genus Nicotiana were tested. The species that developed frenching were *N. alata* Link and Otto, *N. langsdorffii* Schrank, *N. longiflora* Cav., *N. rustica* L., *N. sanderae* Sander, and *N. sylvestris* Spegaz. and Comes. The following species showed no symptoms of frenching: *N. acuminata* Hook., *N. bigelovii* S. Wats., *N. caudigera* Phil., *N. clevelandii* A. Gray, *N. glauca* R. Grah., *N. glutinosa* L., *N. palmeri* A. Gray, *N. paniculata* L., *N. suaveolens* Lehm., *N. tomentosa* Ruiz and Pav., *N. trigonophylla* Dun., and *N. wigandioides* C. Koch and Fint.

Petunia hybrida Vilm. var. Henderson's Giant Empress, *Datura stramonium* L. (Jimson weed), and *Lycopersicon esculentum* Mill. var. Bonny Best

(tomato) were the only other plants in the Solanaceae that showed characteristic symptoms of frenching. The chlorosis in these three species, however, was mild. The solanaceous plants that gave negative results were *Capsicum frutescens* L. (pepper), *Hyoscyamus niger* L. (henbane), *Lycium ferocissimum* Miers., *Nicandra physalodes* (L.) Pers., *Physalis angulata* L. (ground cherry), *Solanum melongena* L. (eggplant), and *Solanum tuberosum* L. (potato).

None of the non-solanaceous plants grown showed any symptom of frenching. The species tested were *Zea mays* L. var. *rugosa* (field corn), *Fagopyrum esculentum* Gaertn. (buckwheat), *Beta vulgaris* L. (beet), *Spinacia oleracea* L. (spinach), *Vicia sativa* L. (vetch), *Glycine max* Merr. (soybean), *Medicago sativa* L. (alfalfa), *Phaseolus vulgaris* L. var. Early Golden Cluster (bean), *Tropaeolum majus* L. (nasturtium), *Gossypium hirsutum* L. (cotton), *Begonia semperflorens* Link and Otto (begonia), *Petroselinum hortense* Hoffm. var. *crispum* (parsley), *Salvia splendens* Ker. (scarlet sage), *Cucumis sativus* L. (cucumber), *Callistephus chinensis* Nees (aster), and *Lactuca sativa* L. var. *crispa* (lettuce).

A number of experiments were made in an endeavor to determine whether frenching is a parasitic, deficiency, or toxicity disease, since it seemed probable that it would fall into one of these three categories. Seedlings of *Nicotiana tabacum* var. Turkish were used as test plants in the experiments described below.

Parasitic Studies

Although Clinton (4), Wolf and Moss (26), and Garner (7) reported that frenching is not a parasitic disease, these investigators did not present the experimental evidence upon which their conclusions were based. Several experiments were made in an attempt to verify this conclusion. A microbiological examination of roots of field-grown healthy and diseased tobacco plants failed to show any association whatsoever between the disease and a pathogen. The following greenhouse experiments substantiated this observation.

When frenched plants were repotted in a mixture of 99 per cent sand and 1 per cent soil, they recovered temporarily within a few days, but became frenched again within 4 weeks. However, similarly diseased plants repotted in 100 per cent sand recovered and remained healthy till the experiment was discontinued. If the disease is due to a parasite, it is difficult to understand why those plants repotted in 100 per cent sand recovered permanently, whereas those placed in 99 per cent sand recovered only temporarily. Sufficient organisms should have been transferred on the roots of the frenched plants in the 100 per cent sand to cause a reoccurrence of the disease, if it is due to a parasite.

In another experiment roots from frenched plants were cut into small pieces and mixed thoroughly with washed sand. Healthy seedlings potted in this mixture and watered with tap water showed no evidence of the disease. When frenched plants were repotted in this mixture, they recovered and remained healthy. When a frenched plant was repotted in a pot of washed sand, in which a healthy plant was already growing, the healthy plant not only remained healthy but the repotted, diseased plant recovered and thereafter showed no symptoms of the disease.

Moreover, soil sterilization did not prevent the development of frenching completely, as would be expected if the disease were caused by a parasite. Soil was sterilized with steam at 15 pounds' pressure in an autoclave for 1 hour on 3 consecutive days. After the 3rd period of sterilizing, samples of soil were plated on agar plates. No growth of any sort developed on the plates within 14 days. Plants potted in this sterilized soil showed the first symptoms of frenching from 2 to 4 weeks later than they did in nonsterilized soil, but eventually all the plants became frenched. This delay in the appearance of symptoms may be explained as follows. Plants in sterilized soil grew faster than similar plants grown in nonsterilized soil, indicating that during the process of sterilization certain organic nitrogenous compounds might have broken down and thereby liberated nitrogen, which became available for plant growth. Therefore, the appearance of frenching was delayed until some of this nitrogen had been used up. The addition of small amounts of nitrogen to soil has been shown to retard the development of frenching. From this experiment and the 3 preceding ones, it appears improbable that frenching is a parasitic disease.

Physiological Studies

Studies were undertaken to determine whether frenching is a deficiency disease or a toxicity disease. Before these studies were carried out, however, it seemed advisable to ascertain the importance of such soil factors as texture, aeration, and reaction in making conditions favorable for the development of frenching.

In the first experiment dealing with soil texture, various amounts of peat and sand were mixed with field soil. Young seedlings were then potted in the mixtures in 4-inch porous clay pots and kept well watered throughout the experiment. It was found that the incidence of frenching in various mixtures of sand and soil was equal to, if not slightly higher than, the incidence of disease in field soil alone. Peat and soil mixtures, on the other hand, lowered the incidence of frenching very noticeably. The number of plants that became frenched decreased as the proportion of peat in the mixture increased. When 50 per cent by volume of the mixture was peat, all plants remained healthy. Neither incidence nor severity of frenching could

be correlated definitely with rapidity of growth. With another set of plants an experiment was made to determine the effect of variations in the amount of watering. When the plants were watered heavily, 75 per cent of them became frenched. With light watering, however, plants of a similar age remained healthy.

Since increasing proportions of peat in soil mixtures decreased and heavy watering increased the incidence of frenching, it was of interest to determine if aeration was a factor in the development of frenching. One set of plants was potted in 100 per cent sand, and a similar set in 99 per cent sand and 1 per cent field soil by volume, in 4-inch pots placed in saucers. The saucers were kept partly filled with water at all times. Within 3 weeks after potting, all the plants in 99 per cent sand and 1 per cent soil became frenched, whereas those potted in sand alone remained healthy even when held under observation in a greenhouse for nearly 4 months. For all practical purposes the aeration and texture of the rooting medium in these two treatments were equal, indicating that frenching, under certain conditions, can develop even with good aeration.

The effect of soil reaction in promoting or retarding the development of frenching is brought out in the following experiment. Various amounts of pulverized lime were thoroughly mixed in a soil that does not produce frenching under ordinary conditions. Young seedlings were potted in these soil mixtures in 4-inch pots placed in saucers that were kept filled with water. The number of plants that developed frenching was observed at weekly intervals. None of the plants became diseased until the end of the 6th week. When the experiment was terminated after 11 weeks, the soil reaction was determined and the green weight of all plants measured. The experimental

TABLE 1.—*Effect of the addition of lime on the development of frenching*

Amount of lime added gm./pot	Number of plants	Number of plants frenched after:						Records after 11 weeks	
		6 weeks	7 weeks	8 weeks	9 weeks	10 weeks	11 weeks	Soil reaction pH	Green weight of plants
0.0	5	0	0	0	0	0	0	7.67	5.0
0.5	5	0	0	0	0	0	0	7.80	7.1
1.0	5	1	1	1	3	5	5	7.90	10.5
1.5	5	2	2	4	4	4	4	8.00	10.0
2.0	5	1	2	2	3	3	3	8.13	14.1
3.0	5	0	0	0	1	1	1	8.23	12.3

data in table 1 show that there was a definite correlation between the hydrogen-ion concentration of the soil and the development of frenching. At pH

7.7 and 7.8 none of the plants became frenched, but at pH 7.9 and 8.0 nearly all the plants developed the disease. Above pH 8.0 a few plants became frenched, but the number decreased as the soil reaction became more alkaline. Even at pH 8.2 the plants made good growth, indicating that the absence of the diseased condition at this reaction was not due to poor growth. The range of soil reaction most favorable for the development of frenching was found to vary somewhat, depending upon other environmental conditions.

The preceding experiments show that soil texture, aeration, and reaction are important secondary factors in the development of frenching. They exert a pronounced action in modifying the incidence and severity of the disease.

Deficiency Studies. In order to determine if frenching might be a deficiency disease, a study was made of the effect on tobacco in sand cultures of a deficiency of the elements known to be essential to plant growth. The nutrient solution was added continuously by means of the constant-drip method of solution renewal (20). Typical symptoms of nitrogen, phosphorus, potassium, calcium, magnesium, and sulphur deficiencies were obtained, but none of these symptoms resembled those characteristic of frenching.

In order to determine the effect of the deficiency of some of these elements when seedlings were grown in soil, a repetition of the above experiment was made, using field soil instead of sand as the growing medium. The nutrient solution was added twice each week, 100 cc. per pot. The plants became frenched when nitrogen or potassium was omitted from the nutrient solution added. In these particular deficiency treatments the plants grew very slowly. Typical symptoms of nitrogen- or potassium-deficiency, in addition to the characteristic symptoms of frenching, were obtained.

These experiments with sand and soil indicate that frenching does not result from a deficiency of any one of the principal essential elements. Since it occurred in soil with the addition of potassium in a nitrogen-deficient nutrient, and also with the addition of nitrogen in a potassium-deficient nutrient, neither a nitrogen nor a potassium deficiency can be considered as the factor causing the disease.

Experiments were then carried out to determine the effect of the elements known to be required for growth in small quantities, such as iron, boron, and manganese. Plants were grown in liquid nutrient cultures in which one of these elements at a time was omitted. None of the symptoms obtained, however, resembled those of frenching. Symptoms other than those of frenching were obtained when an excess of any one of these 3 elements was added. Various amounts of iron, boron, and manganese when added to soil failed to prevent the development of frenching or to bring about recovery when added to soil in which the plants were already frenched.

Since it has been shown by some workers (3, 14, 21) that copper exerts a stimulatory action on plants under certain conditions, tests were made on the effect of copper sulphate on the development of frenching. Table 2

TABLE 2.—*Effect of repeated additions of copper sulphate on the prevention of frenching*

Treatment	$CuSO_4$ (1%) added each week (cc./pot)	Number of plants	Number of plants frenched after:						
			16 days	17 days	20 days	35 days	50 days	65 days	80 days
A	0	5	3	5	5	5	5	5	5
B	5	5	0	3	5	5	3	1	0
C	10	5	0	1	2	2	1	0	0

gives the results of an experiment in which various amounts of $CuSO_4$ were added to field soil. The first treatment was made 5 days after the plants were potted. In group C the $CuSO_4$ exerted some toxic action as the plants were much smaller than those in group B. However, group A and B plants were about equal in size. In group A the symptoms of frenching were very severe. The plants in group B, however, showed only slight chlorosis after 20 days, but by the end of the experiment they appeared to be healthy again. Table 3 shows the effect of adding $CuSO_4$ to plants after

TABLE 3.—*Effect of repeated additions of copper sulphate on the recovery of plants from frenching*

Treatment	$CuSO_4$ (1%) added each week (cc./pot)	Number of plants	Number of plants frenched after:				
			16 days	35 days	50 days	65 days	80 days
D	0	5	5	5	5	5	5
E	5	5	5	5	4	2	0
F	10	5	5	3	2	0	0

they had become frenched. The plants in groups E and F recovered completely, while those in group D showed the rosette condition severely throughout the experiment. The experiments show quite clearly that $CuSO_4$ had an appreciable effect in overcoming frenching and bringing about recovery from the diseased condition. The $CuSO_4$ exerted no measurable change on the hydrogen-ion concentration of the soil.

In order to determine if aluminum had any effect on the development of frenching, various amounts of aluminum sulphate were added to 4-inch pots of field soil. The results obtained when $Al_2(SO_4)_3$ was added at weekly

Photographed by J. A. Carlile

FIG. 2. Plants showing the effect of aluminum sulphate on prevention of frenching. G, check plant on left showing severe frenching; H, plant that received 1 gram $Al_2(SO_4)_3$ showing mild chlorosis; I, plant that received 2 grams $Al_2(SO_4)_3$ showing no frenching. Faint chlorosis appeared on plant I 3 weeks after plants were photographed.

TABLE 4.—*Effect of aluminum sulphate on the development of frenching of tobacco in field soil*

Group	$Al_2(SO_4)_3$ (1 gm./pot) added at weekly intervals for: (weeks)	Number of plants	Number of plants frenched after:						Records after 70 days		
			30 days	35 days	44 days	54 days	60 days	70 days	Soil reaction (pH)	Green weight of plants (gm.)	Remarks
G	0	5	1	5	5	5	5	5	6.80	8.4	Severe rosette
H	1	5			2	5	5	5	6.57	9.3	Chlorosis
I	2	5					1	2	6.45	11.6	Slight chlorosis on 2 plants, rest healthy
J	3	5						1	6.40	7.8	Healthy
K	4	5						0	6.25	7.1	Healthy
L	5	5						0	6.15	5.8	Healthy

intervals to young seedlings grown in soil are recorded in table 4. These data indicate that the doses of $Al_2(SO_4)_3$ added greatly retarded the development of frenching. Figure 2 shows a plant from each of groups G, H, and I, as it appeared 42 days after potting. The extent to which $Al_2(SO_4)_3$ retarded frenching varied with the number of weekly treatments. Thus, with only one treatment, the first appearance of the disease was delayed nearly 2 weeks. With 2 treatments at weekly intervals, disease symptoms did not appear till 4 weeks after those in the check pots. With 3 treatments, only 1 plant out of 5 developed frenching within 10 weeks. The data in table 4 also show that the addition of $Al_2(SO_4)_3$ lowered slightly the reaction of the soil. $Al_2(SO_4)_3$ had only a slight depressing influence on growth. The apparent increase in growth with small amounts of $Al_2(SO_4)_3$, as shown in table 4, was due to the stunting that accompanies the development of frenching.

The addition of $Al_2(SO_4)_3$ was beneficial also in bringing about recovery from the frenched condition. In this experiment, the data from which are given in table 5, all of the plants were frenched before the first addition of $Al_2(SO_4)_3$ was made. Recovery was partial when only 1 or 2 grams of $Al_2(SO_4)_3$ were added per pot, but was complete when 3 grams were added in 1-gram quantities at weekly intervals. The degree to which $Al_2(SO_4)_3$ promoted recovery is shown by the plants in figure 3, photographed 48 days after first treatment. Thus, aluminum was effective not only in preventing the development of frenching, but also in bringing about recovery after the plants had become diseased. Similar results were obtained when $AlCl_3$ was substituted for $Al_2(SO_4)_3$.

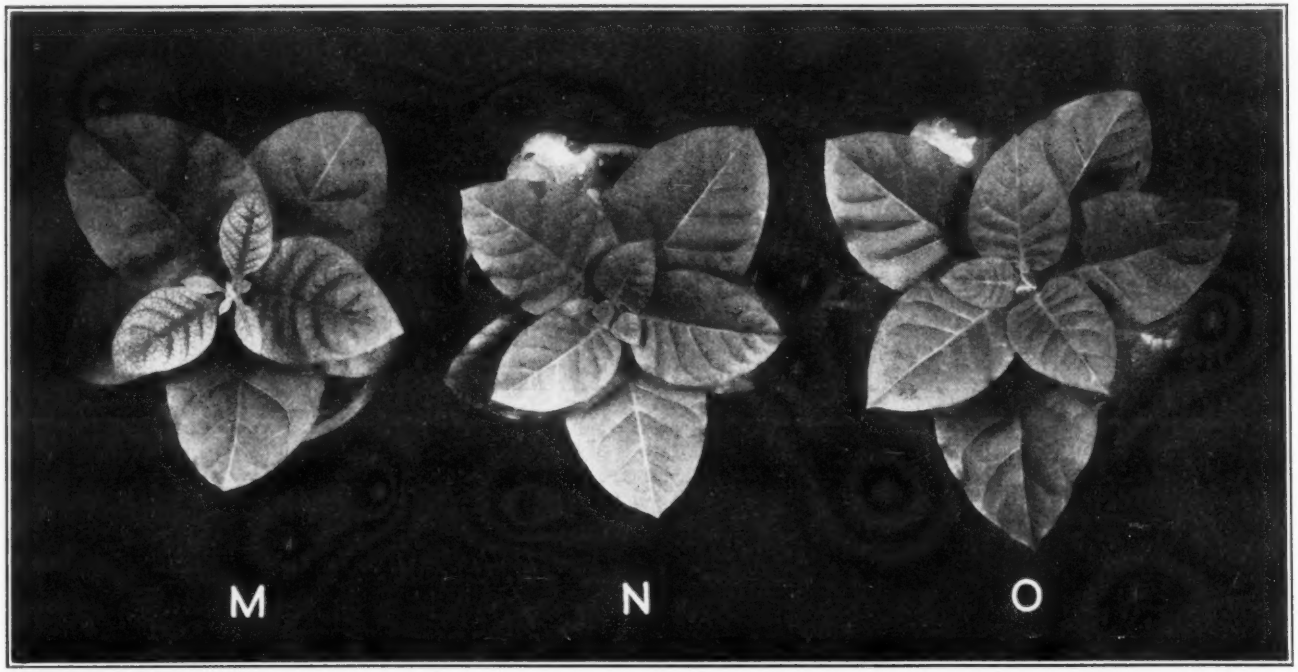
To test the effect of aluminum and copper on the plant itself, young tobacco seedlings were potted in soil in 2-inch pots placed in glass saucers that were kept filled with water. After some 3 weeks all the plants became frenched; the roots by this time were extending down through the hole in the bottom of the pot. The pots were then suspended in glass tumblers. When the extending roots were submerged in a dilute nitrogen solution, the plants recovered. However, when the roots were submerged in a dilute solution of $Al_2(SO_4)_3$ or $CuSO_4$, recovery was only partial.

Inasmuch as frenching failed to develop on plants grown in sand and water cultures without the addition of both copper and aluminum, it is probable that the cause of frenching cannot be considered as a deficiency of either of these two elements, even though they had a pronounced action on the disease. The beneficial effect obtained by adding $Al_2(SO_4)_3$ or $CuSO_4$ to the soil probably was due to some change in the soil complex, rather than to any direct effect of the aluminum or copper on the plant itself.

These experiments on mineral deficiencies indicate definitely that frenching is not caused by a deficiency of nitrogen, phosphorus, potassium, calcium,

TABLE 5.—*Recovery from frenching by the addition of aluminum sulphate to the soil*

Group	$Al_2(SO_4)_3$ (1 gm./pot) added at weekly intervals for: (weeks)	Number of plants	Number of plants frenched after:						Records after 48 days		
			13 days	18 days	23 days	28 days	33 days	48 days	Green weight (gm.)	Soil reaction (pH)	Remarks
M	0	10	10	10	10	10	10	10	4.8	7.10	Severe rosette
N	1	10	10	10	10	10	10	10	6.1	7.05	Chlorosis
O	2	10	9	9	7	5	5	4	6.1	6.85	Faint chlorosis on 4 plants, rest healthy
P	3	10	9	6	4	2	0	0	5.9	6.70	Healthy



Photographed by J. A. Carlile

FIG. 3. Plants showing effect of aluminum sulphate on recovery from frenching. All plants showed frenching before experiment was started. M, plant that received no $Al_2(SO_4)_3$ showing severe frenching; N, plant showing partial recovery after receiving 1 gram $Al_2(SO_4)_3$; O, plant showing complete recovery after receiving 2 grams $Al_2(SO_4)_3$ at weekly intervals.

magnesium, sulphur, iron, boron, or manganese. Since these elements are the only mineral elements commonly considered as essential to plant growth, it is probable that frenching is not directly caused by a deficiency of any mineral nutrient.

Toxicity Studies. Since it was impossible to demonstrate that a deficiency of any of the essential elements is the cause of frenching, the next step was to determine if frenching is a toxicity disease. Experiments were carried out in which such factors as soil complex and mineral nutrition were eliminated as far as possible. In the first experiment seedlings were grown in washed quartz sand in 4-inch pots placed in saucers. One set of seedlings was watered with tap water and a similar set watered with a water extract of field soil. This extract was prepared as follows. Approximately 7 kilograms of soil were placed in a 5-gallon carboy and mixed thoroughly with about 15 liters of distilled water. When the soil had settled to the bottom of the carboy, the liquid was siphoned off and used in watering the plants. The plants watered with soil extract became frenched in about 7 weeks, whereas those watered with tap water were still healthy when the experiment was discontinued 5 weeks later.

In another experiment plants were grown in sand with various proportions of field soil. The experimental data are given in table 6. These

TABLE 6.—*The incidence of frenching in sand mixtures containing very small amounts of field soil*

Proportion of field soil added to sand		Number of plants	Number of plants frenched after:							
Per cent	Parts per thousand		3 weeks	4 weeks	5 weeks	6 weeks	7 weeks	8 weeks	9 weeks	23 weeks
0.0	0.0	5	0	0	0	0	0	0	0	0
0.05	0.5	5	0	0	2	3	3	4	5	
0.5	5.0	5	0	0	1	3	3	5		
0.75	7.5	5	0	0	2	5				
1.0	10.0	5	0	1	2	5				
10.0	100.0	5	4	5						

results show that it is possible to bring about the development of frenching in plants grown in quartz sand by the addition of as little as 1 part of field soil to 2000 parts of sand. Plants grown in sand to which no soil was added remained healthy and showed no symptoms of frenching.

It was later found possible to produce mild symptoms of frenching in plants grown in washed quartz sand without the addition of any soil whatsoever. Plants were grown in quartz sand in 4-inch porous clay pots placed in saucers kept partly filled with tap water from a deep well. At the end

of 5 months the plants, still healthy, were practically dormant, and were replaced by new seedlings. This second set of plants showed the chlorotic stage of frenching within 2 months after potting. These results may be explained as follows. The toxic material probably was present in the tap water in extremely small concentrations. With repeated watering of the plants from the bottom of the pots and continual evaporation of water from the pot and sand, this toxic material gradually accumulated in the sand. By this time the first set of plants had become nearly dormant and showed no evidence of toxicity. However, when new plants were planted in this sand and new growth started, the toxicity was made evident by the typical chlorosis characteristic of frenching in the tip leaves.

The 3 experiments described above would indicate that frenching is a toxicity disease rather than a mineral deficiency disease. The toxic principle was apparently added in the first experiment with the soil extract and in the second experiment with the trace of soil. Further experiments to determine the nature of the toxic principle are under way.

DISCUSSION

Although the experimental evidence presented in this paper indicates that frenching is a toxicity disease, this cannot be considered as conclusively established until the actual cause of the disease has been demonstrated. It is possible that the deficiency of some element, not recognized as essential to growth but nevertheless required in minute amounts and present as an impurity, may be the cause of frenching.

The experimental data have given no indication as to how the plant is affected by the causal agent of frenching. Therefore, it is difficult to explain how nitrogen, aluminum, or copper acts in preventing the development of frenching. These minerals may act directly on a toxic principle or indirectly by affecting some condition necessary for the liberation of such a principle. Soil texture, soil aeration, soil reaction, and mineral plant nutrition were found to be important secondary factors capable of modifying the incidence of the disease.

Further work may lead to a satisfactory explanation of the high susceptibility of certain Nicotiana species and the low susceptibiilty of many other species grown under similar conditions. It is possible that the Nicotiana species affected may be highly sensitive to some toxic element, since it is well known that plants differ in their tolerance of poisons.

The experimental evidence does not permit the formulation of any definite hypothesis as to the cause of frenching. However, the evidence does reveal some facts concerning the nature of the disease and the means by which it may be controlled.

SUMMARY

In the genus Nicotiana frenching developed severely in *N. alata, N. langsdorffii, N. longiflora, N. rustica, N. sanderae, N. sylvestris,* and in 16 varieties of *N. tabacum,* but not in 12 other species of Nicotiana grown under similar conditions. Of the 26 other solanaceous and non-solanaceous species tested, only *Datura stramonium, Lycopersicon esculentum,* and *Petunia hybrida* showed chlorosis characteristic of frenching.

Frenching in tobacco in a greenhouse was controlled by soil composting, the addition of peat, repeated applications of a nitrogenous fertilizer, and by several applications of a dilute solution of copper sulphate or aluminum sulphate.

No association was found between frenching and any pathogenic organism.

Experiments on the deficiency of each of the elements essential to plant growth failed to produce symptoms that resembled those of frenching.

Frenching was produced in plants grown in sand by the addition at daily intervals of a water extract of field soil. It also was produced by adding as little as 1 part of field soil to 2000 parts of sand. In young seedlings, in sand, it was produced by the addition of tap water from a deep well, over a long period of time.

The experimental evidence indicates that frenching probably is not a mineral-deficiency disease, but a toxicity disease produced by some toxic principle that is present in certain soils and that exerts its toxic action only under definite environmental conditions.

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A STUDY OF COALESCING HAPLOID PUSTULES IN PUCCINIA HELIANTHI[1]

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(Accepted for publication February 18, 1935)

INTRODUCTION

It was shown by Craigie (5) that, in *Puccinia helianthi* Schw., when two haploid pustules of opposite sex coalesce, aecia develop in the compound pustule thus formed. He drew the theoretical deduction that hyphal fusions occur between the two mycelia and give rise to the diploid condition in which a nucleus of one sex is associated in conjugate relationship with a nucleus of the opposite sex. Somewhat later, however, he (6) discovered that the transfer of pycnial nectar from a haploid pustule of one sex to the pycnia of a haploid pustule of the opposite sex initiated the production of aecia in the pustule receiving the nectar.

The latter discovery raises the question as to whether or not the process of diploidisation can be initiated in *Puccinia helianthi,* or in any other similar heterothallic rust, by hyphal fusions between two haploid mycelia of opposite sex. In Craigie's early experiments, there was the possibility that the coalescence of pustules of opposite sex might have afforded an opportunity for an intermingling of their nectar. If that happened, the aecia might have arisen as a result of the functioning of the pycniospores, rather than as a result of hyphal fusions. To obtain further information concerning the point in question, an experiment was carried out in which precautions were taken to prevent any interflow of the pycnial nectar of coalescing pustules, or any accidental transference of nectar from one pustule to another.

In the Hymenomycetes, it is now well-established that the diplophase follows the union of paired haploid mycelia of opposite sex; and Buller (3 and 4) has shown that, when a haploid and a diploid culture of *Coprinus lagopus* coalesce, the mycelium of the former becomes diploidised. A similar result was obtained by Dickson (7) with *Coprinus sphaerosporus.*

Brown (2) found that when a haploid and a diploid (uredial) pustule of *Puccinia helianthi* coalesced, the haploid pustule became diploid and produced aecia. As pycnia are entirely absent from uredial pustules and as precautions were taken in the experiment to prevent any fortuitous transference by insects of pycnial nectar from one haploid pustule to

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another, no other conclusion seems possible but that the diplophase arose as a result of the interaction of the haploid and diploid mycelia. In a cytological study of heterothallism in *Puccinia coronata* Corda, Allen (1) mentions finding a hyphal fusion (illustrated in Plate 11, B) that she suggests may indicate the origin of the diplophase by hyphal fusion between haploid mycelia.

EXPERIMENTAL METHODS AND RESULTS

Haploid pustules of *Puccinia helianthi* were obtained by sowing sporidia from germinating teliospores on seedling plants of the cultivated sunflower, *Helianthus annuus L.* When the pustules appeared a careful inspection of them was made to distinguish as far as possible those of monosporidial origin from those of bisporidial origin. Such an inspection is fairly successful if made when the tiny pustule first excretes a minute globule of nectar. Among those pustules that appeared to be monosporidial, only those were selected that occurred in pairs, and among the pairs only those in which the two components of the pair were separated from each other by a distance of from 2 to 4 millimeters. Care was taken to protect the plants bearing the pustules from any agency that might accidentally intermix the pycnial nectar.

As a further precaution that all the pustules used in the experiment were haploid, the pustules were allowed to develop until they were 2 weeks or more old. If any individual pustule was of bisporidial origin and consisted of mycelia of opposite sex, it would most probably have produced aecia by the time it had reached this age. Only those pairs in which the two individual pustules were at this time still separated by a narrow strip of green tissue, or slightly chlorotic tissue in which no pycnia had yet developed, were included in the experiment proper.

In order to prevent the spontaneous intermixing of the nectar of each pair of pustules when later the two components would coalesce to form a compound pustule, a barrier was placed on the upper leaf surface between the two pustules of each pair. For this purpose, Eastman's Opaque No. 1 preparation was found quite satisfactory. It adhered readily to the leaf, dried quickly, and caused no perceptible injury. The barrier, or wall, was placed at right angles to a line passing through the centres of the two pustules comprising each pair and extended sufficiently far on either side of this line to assure its always exceeding any diameter that the pustules might later attain. The experiment was carried out during the winter of 1933–34. At this season of the year in Winnipeg the days are short and the light intensity is rather low, conditions under which the production of nectar is scanty and confined mostly to the central part of the pustule. The plants at this time of the year are almost free from insects. Hence

the possibility of any intermixing of pycnial nectar was practically eliminated.

A total of 288 pairs of pustules were included in the experiment. The 2 pustules of each pair were separated by a barrier of Opaque, as described above. Finally, they coalesced. The barrier appeared to have no adverse effect on the growth of the pustules: the radial expansion of the pustules, as observed on the under side of the leaves, was quite uniform. Of the 288 compound pustules that arose through the coalescence of the individual pairs of simple pustules, 110 produced aecia.

In the compound pustule that produced aecia, it almost invariably happened that aecia appeared in one component first and afterwards in the other component, aecial production being usually completed in the one before it became evident in the other (Fig. 1, A). In both components,

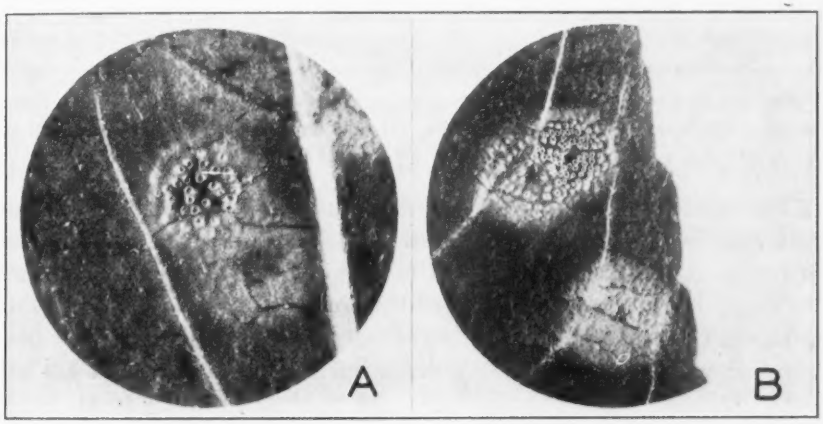

FIG. 1. A. Compound pustules of *Puccinia helianthi*. Aecia have formed in one component, but not in the other. ×5. B. Simple and compound pustules of *P. helianthi*. In one component of the compound pustule, the aecia are well developed, whereas in the other, the aecia are only beginning to appear. ×3.

aecia developed first, although at different times, in the areas adjacent to the line of fusion and latest in the areas most remote from that line. Figure 1, B shows that in one of the components the production of aecia is about completed, and that in the other component the development of aecia has progressed farthest near the line of fusion. There can be little possible doubt that, under the conditions of this experiment, the diploidisation process originates in the areas where the hyphae of the two mycelia first intermingle and that this process, as a rule, runs its course in one component before it makes any noticeable progress in the other.

The progressive diploidisation of one component of a compound pustule,

beginning in the region of coalescence and extending outward from this region to the opposite side of the component is further illustrated in figure 2, A, which shows a photomicrograph of a portion of a section cut parallel

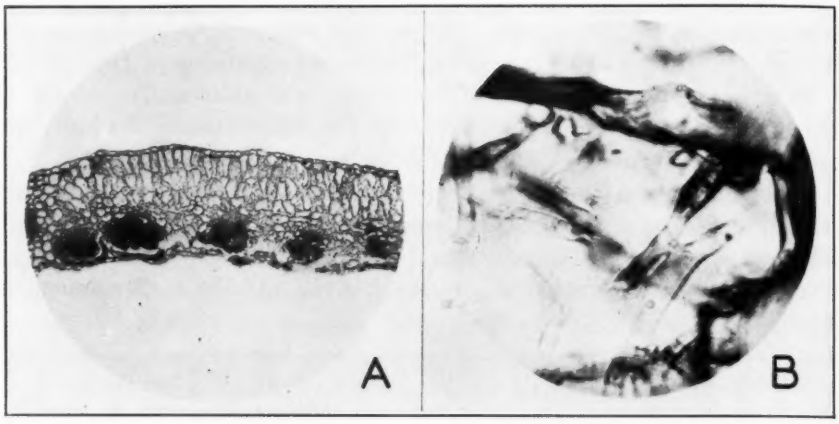

FIG. 2. A. Cross section through one component of a compound pustule of *Puccinia helianthi*. Explanation in the text. ×25. B. Hyphal fusion found in the region of coalescence of a compound pustule of *P. helianthi*. × about 460.

to a line joining the centres of two pustules that had coalesced. A macroscopic examination of the under surface of the compound pustule, made just before the material was placed in a fixing solution, showed that aecia were beginning to develop in one of the components but not in the other. The region of coalescence is just to the left of the part shown in the photograph. It will be seen that the 2 neighbouring aecia on the left are about equally well developed and that both are producing chains of aeciospores (represented by the deeply-stained areas). In the third aecium the left-hand portion has become diploid and this portion is proceeding with the formation of aeciospores, although progress in this operation is less advanced than in the 2 aecia on the left. The remainder of the third aecium is still haploid, as are also the 2 aecial primordia still further to the right.

In view of the precautions taken in this experiment to guard against the transference of pycnial nectar from one pustule to another, the results obtained indicate that when 2 haploid pustules of opposite sex coalesce, they are able to diploidise each other without the intervention of pycniospores. It would appear that the interchange of nuclei must take place through hyphal fusions. In the region of coalescence, hyphal fusions have been found in compound pustules that were beginning to produce aecia. How many may occur in one compound pustule is not known, but at least one has been found in each of several compound pustules that were sectioned. Such a hyphal fusion is shown in figure 2, B. But no definite evidence

of nuclear migration was observed in any of the different fusions examined, and moreover, it was impossible to determine whether the fusions had occurred between 2 hyphae of the same mycelium, or between a hypha belonging to one mycelium and one belonging to the other mycelium.

DISCUSSION

The occurrence of aecia in one component of a compound pustule earlier than in the other component is a phenomenon that lends itself to speculation, but one for which it would seem difficult to determine the exact cause. As it occurs with such marked regularity, there must be some cause of its occurrence. One would suspect that when the two mycelia meet there would be ample opportunity for several, or even many, hyphal fusions to occur, and, therefore, opportunity for the diploidisation of the 2 components to take place simultaneously. This however very rarely happened. It may be that it is just a matter of chance as to which component first becomes diploidised and produces aecia. If that is the case, and on the assumption that only one hyphal fusion takes place, it may be supposed that a nucleus of one mycelium on entering the other mycelium proceeds rapidly to diploidise the latter mycelium. The formation of aecia follows close upon the advance (across the component) of the diploid condition. Such a sequence of events may so affect the physiological balance of the mycelium undergoing diploidisation that, until an approach to physiological equilibrium (complete or nearly complete diploidisation) in it is restored, no nucleus can pass over to the other mycelium to initiate the diploidisation of it. On the assumpton that 2 or more hyphal fusions occur and that 2 or more nuclei pass out from their own mycelium into the other mycelium, the movement must be in one direction first, namely, toward the mycelium that first becomes diploidised, and later towards the other mycelium; for, if nuclei passed in both directions, it is difficult to see why both mycelia do not become diploidised simultaneously.

On the other hand, it may be that it is not a matter of chance as to which component becomes diploidised first. There is the possibility that in compound pustules of which the components are of opposite sex, diploidisation regularly occurs first in those components belonging to one particular sex, for instance, in all the (+) components. Dickson (7) found that, when he mated 8 haploid strains of *Coprinus sphaerosporus*, not only did paired mycelia of opposite sex on uniting diploidise each other, but that, in general, the diploidisation was effected several days earlier in one member of each reacting pair than in the other. Also, he found that the strains first diploidised belonged to one sex, and could be distinguished from the other strains by their thinner aerial mycelium and their slower growth rate. The work reported in the present paper was done prior to the publication of

Dickson's paper, and no particular attention was paid to the rate of increase in pustule size, the character that in *Puccinia helianthi* approaches most nearly to, but probably cannot be considered a substitute for, rate of mycelial growth; but if the components that first produced aecia were regularly of less diameter than their respective mates, it is unlikely, although not impossible, that this difference passed unnoticed. Of course, the point can be decided only by someone making the necessary measurements.

Unless it be found that haploid pustules of one sex consistently increase in size more rapidly than haploid pustules of the opposite sex, the determination of which pustules are of one sex and which of the opposite sex will prove a very difficult matter, as in all other macroscopic characteristics pustules of both sexes are, as far as known, identical; and biochemical methods of differentiation would encounter very great difficulties, owing to the limited size of the pustules and to the fact that the organism can develop only in intimate association with the tissues of the host.

SUMMARY

Evidence is presented indicating that, in *Puccinia helianthi* Schw., when 2 haploid pustules of opposite sex coalesce to form 1 compound pustule, diploidisation of the mycelia of the components of the compound pustule can be effected without the intervention of pycniospores. The diploidisation process is initiated, apparently, by hyphal fusions and it is completed, or almost completed, in one component before it is begun in the other component.

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STUDIES ON SEVEN DIFFERENTIATING CHARACTERISTICS OF TWO PHYSIOLOGIC FORMS OF TILLETIA TRITICI[1]

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(Accepted for publication March 4, 1935)

INTRODUCTION

Physiologic forms of *Tilletia tritici* (Bjerk.) Wint. are identified and separated primarily on the basis of their pathogenic properties. This, however, does not necessarily mean that they are alike in all other respects. Differences between physiologic forms of *T. tritici* with respect to smut-ball type, prominence of the spore-wall reticulations, and degree of stunting of the plants have been noted by various workers, while other reports (4, 6) indicate that strains of the bunt organisms do not differ consistently in cultural characteristics. Bressman (2), Smith (11), and Brentzel (1) have reported differences in physiologic forms of *T. tritici* with respect to type of smut balls produced, and Rodenhiser (9) found that certain forms of the bunt organisms stunt the plants more than do others. It is evident, therefore, that physiologic forms of *T. tritici* may differ in several respects other than pathogenicity. As yet, however, little consideration has been given to the significance of these differences as criteria in establishing the identity of physiologic forms of *T. tritici*.

In 1933 the writer (5) reported that 2 physiologic forms of *Tilletia tritici* on the variety Hohenheimer (C. I. 11458) were distinguishable on the basis of pathogenicity, spore-ball size and shape, spore-wall reticulations, or rate of chlamydospore germination. Further studies have shown that these 2 forms of *T. tritici* differ also in certain other characteristics. The difference in rate of chlamydospore germination reported previously has been less constant than preliminary tests had indicated. Therefore, it is not included in this discussion.

RESULTS

In the physiologic-form smut nursery at Pullman, Washington, in 1931, a form of *Tilletia tritici* collected at Pullman produced large, elongate smut balls on the variety Hohenheimer, while another form, from Lind, Wash., produced small, somewhat rounded smut balls on the same variety (Fig. 1). According to the results in 1931, there was no significant difference between the pathogenic properties of these 2 forms of *T. tritici*. Nevertheless, they were considered as distinctly different strains on the

[1] Cooperative investigations between the Division of Cereal Crops and Diseases, Bureau of Plant Industry, United States Department of Agriculture, and the Washington, Oregon, and Idaho Agricultural Experiment Stations.

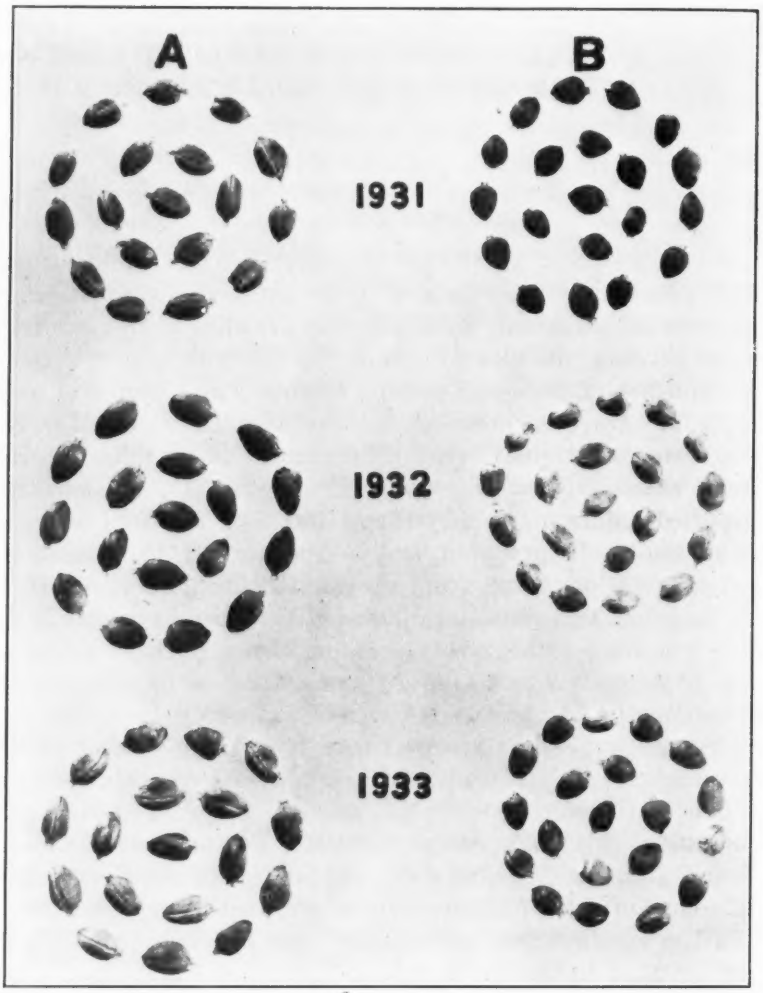

FIG. 1. Types of smut balls produced on Hohenheimer by 2 physiologic forms of *Tilletia tritici*. A. Pullman form. B. Lind form.

sole basis of difference in smut-ball type. Additional tests were made at Pullman, Wash., and at Pendleton and Corvallis, Oreg., to determine whether this difference would remain constant over a period of years and under different environmental conditions. It was considered probable, also, that a difference in pathogenicity might be exhibited in these more extensive tests. The results of 3 years' tests, 1932–34, indicate clearly that the 2 forms are distinctly different pathogenically (Table 1). Based on a 3-year average, the Pullman form produced 33.0 per cent smut on Hohenheimer as compared to 81.0 per cent produced by the Lind form on

TABLE 1.—*Differentiating characters of 2 physiologic forms of Tilletia tritici on the variety Hohenheimer*

Form	Smut	Smut-ball size and shape	Spore-wall reticulations	Mean diameter of spores	Mean length of promycelia	Mean height of culms		Nuclear behavior
						Smut-free	Smutted	
	Per cent			μ	μ	*Cm.*[a]	*Cm.*[a]	
Pullman, Wash.	33.0	Large and elongate	Faint	17.85 ± 0.03	72.62 ± 1.60	118.48 ± 0.44	99.80 ± 0.52	Nuclei scattered
Lind, Wash.	81.0	Small and rounded	Prominent	19.31 ± 0.04	21.45 ± 0.39	118.48 ± 0.44	92.84 ± 0.43	Nuclei in a group

[a] Measurements on the height of culms were made on the variety Hosar.

the same variety. Approximately the same difference in pathogenicity of the 2 forms was apparent on Hosar (C. I. 10067), but the other varieties tested, except Hybrid 128 (C. I. 4512), were highly resistant. The differences in smut-ball type were constant over a 4-year period, 1931–34, as shown for the first 3 years at Pullman, in figure 1. Similar differences in pathogenicity and spore-ball type were observed at Pendleton, Oregon.

The results of studies to ascertain in how many ways these 2 Hohenheimer forms of *Tilletia tritici* differ are summarized in table 1. All studies were made with material taken from Hohenheimer, except for the study of the relative degree of stunting of the plants, in which the variety Hosar was used. It was established, as indicated in table 1, that there are 7 ways in which these 2 forms may be distinguished from each other. As reported in 1933 (5), there is, in addition to pathogenicity and spore-ball characteristics, a distinct difference in the prominence of the spore-wall reticulations. The reticulations on the chlamydospores of the Pullman form are relatively faint or inconspicuous in contrast to the very prominently reticulated walls of the spores of the Lind form, which give the spores of this form a somewhat spiny appearance. Brentzel (1) has reported similar observations on 2 forms of *T. tritici* on Ceres wheat.

A difference in the size of the chlamydospores of the two forms of *Tilletia tritici* on Hohenheimer also was established. Measurements were made of 252 chlamydospores of each form, and it was found that the mean diameter of the spores of the Pullman form averaged $17.85 \pm 0.03\ \mu$ as compared to $19.31 \pm 0.04\ \mu$ for the Lind form (Table 1). These figures show a significant difference of $1.46 \pm 0.05\ \mu$ in the diameter of the chlamydospores of the 2 forms. Similarly, measurements were made of 100 promycelia of each of the 2 forms, and it was found that the mean length of the promycelia of the Pullman form was $72.62 \pm 1.60\ \mu$ as compared to $21.45 \pm .39\ \mu$ for the promycelia of the Lind form. This shows a difference in length of the promycelia of $51.17 \pm 1.65\ \mu$, which is 31 times as great as the probable error of the difference. The measurements were made from the points of attachment of the promycelium to the chlamydospore and the primary sporidia to the promycelial tip. It also was found that there was a difference in the degree of stunting of the plant caused by these 2 forms. The mean height of 92 smut-free culms was 118.48 ± 0.44 cm., as compared to a mean height of 99.80 ± 0.52 cm. for 80 culms infected with the Pullman form and 92.84 ± 0.43 cm. for 176 culms infected with the Lind form. These figures show that there is a definite stunting of the plants by both forms, the difference in height between the smut-free culms and culms smutted with the Pullman form being 17.41 ± 1.68 cm., while the difference in height between the smut-free culms and culms smutted with the Lind form was even greater, 24.31 ± 1.61 cm. The difference

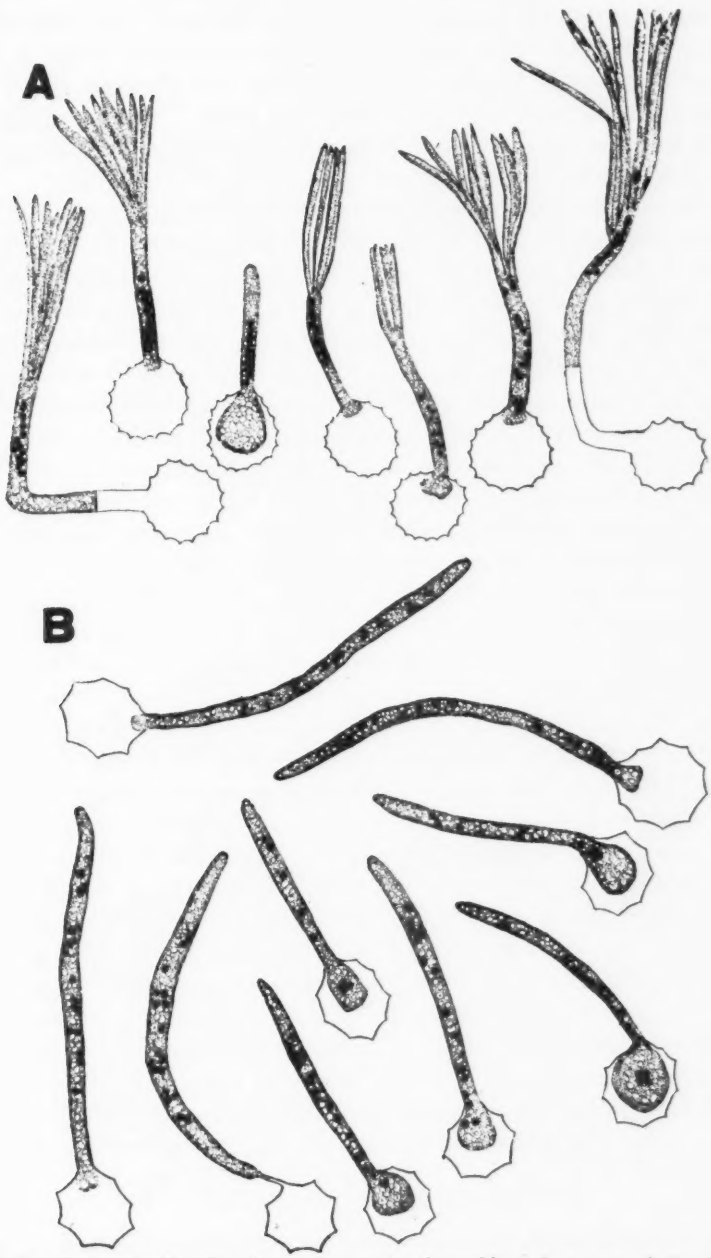

FIG. 2. Camera-lucida drawings of germinating chlamydospores of 2 physiologic forms of *Tilletia tritici*, showing the difference in nuclear distribution in the promycelium. A. Lind form. B. Pullman form. ×830.

in the height of the culms infected with the Pullman form and the Lind form was 6.89 ± 0.68 cm., showing that the Lind form causes a greater degree of stunting in the variety Hosar than the Pullman form. Field observations indicate rather definitely that these 2 forms of *T. tritici* differ in this respect also on Hohenheimer, but actual measurements on this variety have not been made.

A difference in the nuclear behavior at the time of germination of the chlamydospores of the 2 forms of *Tilletia tritici* also was established. As indicated in table 1, the nuclei of the Pullman form were scattered, while those of the Lind form remained in a group. Aside from its significance as a differentiating character between these 2 forms of *T. tritici,* this difference in nuclear phenomenon may help to explain the conflicting view-points on nuclear behavior in *Tilletia spp.* expressed by earlier workers. Dastur (3) observed that the undivided spore nucleus of *T. tritici* passed into the single-cell promycelium before the first and subsequent divisions occurred. On the other hand, Paravicini (7) had previously reported that the first division takes place within the spore, and one nucleus passes into the promycelium, the other remaining in the spore. Later, Rawitscher (8) maintained that all nuclear divisions take place within the spore during germination and that, when the promycelium is produced, as many as 16 nuclei may migrate into it. The type of nuclear behavior observed in the 2 physiologic forms of *T. tritici* described in this paper is similar to that observed by Rawitscher (8). All divisions of the nuclei take place within the spore and later the nuclei migrate into the promycelium. However, the distribution of the nuclei in the spore and promycelium is not the same in the 2 forms. As pointed out previously, the nuclei in the Pullman form were scattered, while those in the Lind form were grouped. This difference was apparent while the nuclei were still in the spore, as shown in figure 2, and it continued to persist during and after the process of migration into the promycelium, as shown in figure 3. Certain exceptions to the general rule were observed, and some of these exceptions are shown in figures 2 and 3. Exceptions are expected, however, but they do not invalidate the general rule. In view of the difference in the nuclear behavior in the 2 forms of *T. tritici* described herein, it seems very probable that the opposing view-points of Paravicini (7), Dastur (3), and Rawitscher (8) were based on studies of different physiologic forms.

SUMMARY AND CONCLUSIONS

Seven differentiating characteristics were established between 2 physiologic forms of *Tilletia tritici* that attack the variety Hohenheimer (C. I. 11458). Aside from differing in pathogenic properties on Hohenheimer, the 2 forms differ also in type of smut ball, prominence of the spore-wall

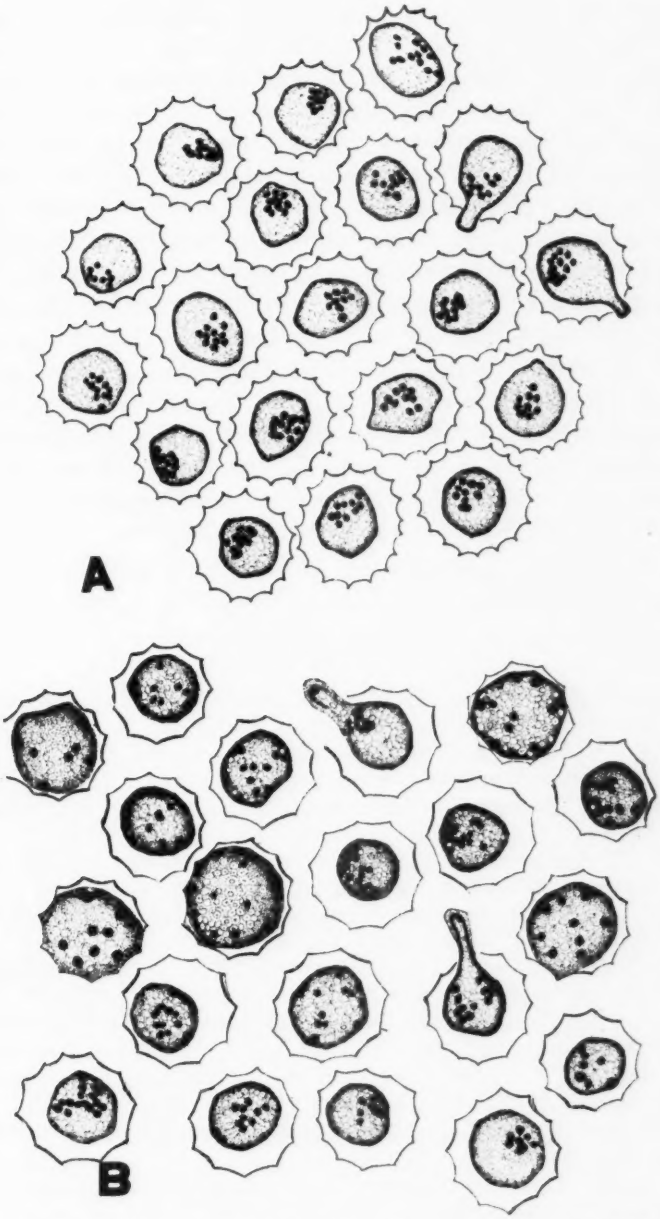

FIG. 3. Camera-lucida drawings of chlamydospores of 2 physiologic forms of *Tilletia tritici*, showing the difference in nuclear arrangement in early stages of germination. A. Lind form. B. Pullman form. ×1850.

reticulations, size of the spores, length of the promycelia, and the nuclear behavior during germination of the chlamydospores. On the variety Hosar (C. I. 10067), they differ also in the degree of stunting of the host plants. This is the largest number of characteristics in which 2 physiologic forms of *T. tritici* have been found to differ. Three of these differences—size of chlamydospores, length of promycelium, and nuclear behavior—apparently have not been reported previously. While it is recognized that, from an economic point of view, a difference in the pathogenicity of physiologic *forms of T. tritici* and *T. levis* is more important than other differences, it also is recognized that, in view of Smith's (10) results, pathogenic properties may vary under different environmental conditions. For this reason, it cannot be expected that the pathogenic reaction of physiologic forms of these organisms will be the same in every locality. Therefore, in view of the great need for a uniform system of identifying and numbering physiologic forms of *T. tritici* and *T. levis*, perhaps it would be wise to take into account, because of their possible usefulness in establishing such a system, differences (other than pathogenicity) between forms.

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EFFECT OF CROWN GALL, HAIRY ROOT, AND WOOLLY APHIDS ON APPLE TREES IN THE ORCHARD

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(Accepted for publication February 13, 1935)

The losses to individual apple nurseries in Tennessee during the early twenties, due to the culling of trees on which had developed crown gall, hairy root, and woolly-aphid knots, often amounted to 50 per cent—sometimes running as high as 90 per cent—of the total number of trees produced. The situation called for two lines of study: one to find, as quickly as possible, effective, practical methods of control; the other to determine by actual orchard tests just how important it is to avoid, in Tennessee, the planting of affected trees. This paper is a report of the latter study, in which tests were made of the effects of the troubles on trees in the orchard under Tennessee conditions.

Such tests were important, first, because most workers, especially in the northeastern States, were convinced that crown gall was of no significance in the orchard; and, second, because many of our nurserymen reported apparently definite observations, and what appeared to be special tests, showing that culled nursery trees, seriously affected with one of the troubles in question, produced as well in the orchard as any so-called healthy trees. The nurserymen did not believe it necessary to destroy the culled trees.

To obtain experimental data on this much disputed question that would apply to conditions in Tennessee, a test orchard was set out on the West Tennessee Experiment Station farm, at Jackson, early in the spring of 1923. The location had previously been set out to apple trees, most of which died or became undesirable, and the plot, therefore, was cleared prior to the setting of the test trees. Four times as many trees were set out as were expected to remain permanently in the orchard.

Twelve hundred trees were planted—the number of each variety being as follows:

Black Ben Davis	135
Delicious	80
Early Harvest	165
Horse	55
Kinnard	105
Mammoth Black Twig	80
Red June	70
Rome	55
Stayman	200
Wealthy	40

Winesap	120
Yellow Transparent	40
York Imperial	55
Total	1200

The trees were set in the rows according to variety. At the time of planting, 19.8 per cent had large crown galls, 11.5 per cent medium-size, and 9.5 per cent small crown galls; 20.1 per cent had extensive and 8.2 per cent slight development of hairy root; 9.3 per cent had extensive and 8.5 per cent slight development of woolly-aphid knots; and 13.1 per cent were apparently healthy. So far as diseases and injuries were concerned, the trees were planted at random. They were neither fumigated nor disinfected in any manner.

In February, 1924, one year after planting, 55 trees in permanent locations were found dead and were replaced with apparently healthy ones. The distribution of the dead trees according to their condition at planting time is shown in table 1.

TABLE 1.—*Distribution of dead trees in permanent locations, February, 1924, according to their condition at planting time*

Condition of tree at planting time, spring of 1923	Dead trees in permanent locations, February, 1924	
	Number	*Per cent*
Crown gall, large	18	32.6
" " medium	3	5.5
" " small	3	5.5
Hairy root, extensive	6	10.9
" " slight	5	9.1
Woolly-aphid knots, extensive	14	25.4
" " " slight	3	5.5
Apparently healthy	3	5.5

The number of trees recorded in table 1 is very small, and most of the figures, therefore, have little significance. They show, however, that the death rate was rather high—significantly so, it appears—among the trees that were severely affected with woolly aphids at planting time or had large crown galls, compared with those that were affected slightly or not at all.

In December, 1925, 3 seasons after the test trees were set out in the orchard, 595 of them, or nearly half the total number, were dug up in systematic order and their condition recorded, with the results shown in table 2.

With the exception of the data for the trees originally having medium-size crown galls, the figures presented in table 2 indicate that the death rate and health condition of the trees were in correlation with the condition

TABLE 2.—*Condition of trees dug up in December, 1925, three seasons after being set in test orchard, classified according to their condition at planting time*

Condition of tree at planting time, spring of 1923	Number of trees dug up	Condition of trees at digging time, December, 1925			
		Dead	With large crown gall	With poor or rotted roots	Poor roots and dead combined
		Per cent	*Per cent*	*Per cent*	*Per cent*
Crown gall, large	123	34.1	35.8	5.7	39.8
" " medium	66	9.1	37.9	6.1	15.2
" " small	50	18.0	28.0	14.0	32.0
Hairy root, extensive	134	18.6	31.4	17.9	36.5
" " slight	49	14.3	28.6	18.3	32.6
Woolly-aphid knots, extensive	33	54.6	12.1	3.1	57.7
" " " slight	79	20.2	15.2	7.6	27.6
Apparently healthy	61	4.9	21.3	6.6	11.5

of the trees at planting time. The highest death rate, 54.6 per cent, was for the trees originally showing extensive development of woolly-aphid knots;[1] and the next highest, 34.1 per cent, for those originally having large crown gall. The lowest death rate, 4.9 per cent, was for the trees that were apparently healthy at planting time. The remaining classes, except one originally with medium-sized crown gall, occupy about the same position—an intermediate between that of the trees extensively affected with woolly-aphid knots and with large crown gall, on the one hand, and trees apparently healthy on the other. The figures in the last column, showing percentage of the dead trees and those with poor or rotted roots combined, also appear to be significant. These figures indicate that the number of dead trees and trees with poor or rotted roots is in direct proportion to the degree of development of any of the troubles affecting the trees at planting time. The number is the highest for extensive woolly-aphid infestation, followed in order by large crown gall, and extensive hairy root development. The death rate for the trees apparently healthy at setting time is the lowest, and for all others—again except the group originally with medium-size crown gall —occupying an intermdiate one.

The exceptionally low figures for dead and poorly rooted trees in the medium-size crown-gall group can be explained by the chance location of most of the trees in soil unfavorable for rapid dying or for severe development of the trouble, or by the possibility that in this group were placed many trees not affected with true crown gall but with moderately developed

[1] If the trees with woolly-aphid knots had been effectively fumigated before planting, the results might have been different.

callus overgrowths. This might actually have happened, because at planting time no sharp distinction was made in the grading between crown gall and callus overgrowth, though the difference was known to the graders and was most likely observed, at least in the grading of trees with large galls. In any case, the exception shows that under the conditions of the experiment no special significance can be attached to small differences in all of the figures or to an isolated record, even with a markedly different figure.

In December, 1928, 6 seasons after the trees were planted, 157 of them were dug up, again in systematic order, and their general condition recorded, as shown in table 3.

TABLE 3.—*Condition of trees dug up in December, 1928, six seasons after being set in test orchard, classified according to their condition at planting time*

Condition of tree at planting time, spring of 1923	Number of trees dug up	Condition of trees at digging time, December, 1928				
		Very good	Good	Poor[a]	Dead	Dead and poor
		Per cent	*Per cent*	*Per cent*	*Per cent*	*Per cent*
Crown gall, large	31	19.3	19.3	35.5	25.9	61.4
" " medium	25	20.0	44.0	28.0	8.0	36.0
" " small	30	13.3	50.0	23.4	13.3	36.7
Hairy root, extensive	29	13.8	51.7	13.8	20.7	34.5
" " slight	10	0.0	70.0	30.0	0.0	30.0
Woolly-aphid knots, extensive	12	25.0	8.3	16.7	50.0	66.7
Woolly-aphid knots, slight	7	43.4	28.6	14.0	14.0	28.0
Apparently healthy	13	61.5	23.1	7.7	7.7	15.4

[a] Trees with root system in such poor condition that they were expected to be dead within 2 or 3 years.

While the figures in table 3 are small and no special significance can be attached to any individual case, the general trend is indicative of certain correlations. It seems clear, for example, that the number of dead and "poor" trees combined is around 4 times as great for the trees originally with extensive woolly-aphid knots and with large crown galls, and twice as great for the other classes of affected trees, as for the originally healthy trees.

The trees that were not dug up in 1925 and 1928 were those planted mostly in permanent locations. These were left undisturbed except that now and then dead ones were replanted. The first record of such dead, replanted trees was made in 1924 and has been already presented here. The remaining records have not yet been completely analyzed, hence they are not available

for presentation at this time. The figures that are available, however, indicate the same trend as those discussed, namely, that the highest death rate was among the trees originally with extensive development of woolly-aphid knots and with large crown galls, and the lowest among the trees apparently healthy at planting time.

In conclusion, it may be stated that the test seems to indicate clearly that the death rate of apple trees in the orchard is correlated with, and proportional to, the development of crown gall, woolly-aphid knots, and, apparently, hairy root, on the trees used for the planting. The test shows clearly that it is not safe to plant such trees in the orchard. It must be understood, however, that the trees were neither fumigated nor disinfected in any manner before they were planted.

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DATES OF PRODUCTION OF THE DIFFERENT SPORE STAGES OF CRONARTIUM RIBICOLA IN THE PACIFIC NORTHWEST

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(Accepted for publication February 26, 1935)

White pine blister rust (*Cronartium ribicola* Fisch.) was first discovered in western North America in the vicinity of Vancouver, British Columbia, in the fall of 1921.[2] Following this discovery, investigations of the disease have been conducted annually in the West since 1922 by the Division of Forest Pathology, Portland, Oregon, for the purpose of accumulating facts on the behavior of the rust under the new host and environmental conditions pertinent to the development of control measures. One phase of these large-scale studies has been to secure data annually on the date of occurrence of the various spore stages in the life history of the rust on white pines and ribes.[3] These data, which are herein presented in tabular form (Tables 1 and 2), show some of the more important dates in the development of the white pine blister rust on the two different hosts, and their variation according to season and locality.

In tables 1 and 2 it will be noted that British Columbia has been first broadly divided into 2 regions—coastal and interior. Subdivisions of these 2 regions have then been made and the data grouped thereunder. The map (Fig. 1) shows regional divisions, subdivisions, and localities referred to in the tables. The dry belt includes that part of the interior region outside the range of *Pinus monticola* Dougl. The Canadian Pacific Railway has been used as the dividing line between the southern and northern dry-belt sections. All other regional subdivisions or localities mentioned are self-explanatory on the map. All subdvisions are based upon climatic, altitudinal, and vegetational differences between the particular sections of country in question. No data were obtained in the higher elevations of the

[1] Assistant Pathologists, Division of Forest Pathology, U. S. Bureau of Plant Industry, Portland, Oregon. The writers were assisted in the collection of data used in this paper by H. G. Lachmund and T. S. Buchanan, of the Division of Forest Pathology; by C. N. Partington, A. A. McCready, and W. F. Cummins, formerly of that Division; and by several members of the Division of Plant Disease Control, Spokane, Washington.

[2] Davidson, A. T. Report of the Proceedings, Third Western White Pine Blister Rust Conference, Portland, Oregon, Nov. 22, 23, 1923 (Mimeographed).

[3] The genus name Ribes is used in this paper to include both currants and gooseberries.

TABLE 1.—*Dates of aecial and pycnial development on white pines in different regions*[a]

Development on pines	Year	BRITISH COLUMBIA — Coastal Region: Near sea level Elevation 0-200'	Coastal Region: Garibaldi (Mile 22, PGE Ry.) Elevation 1100'	Coastal Region: Mile 72 PGE Ry. Elevation 1300'	Coastal Region: Prohibition Claim Plot and Tenquil Elevation 3000-5400'	Interior: Range of *Pinus monticola* Elevation 1150-2500'	UNITED STATES — Washington: Western Wash. Range of *Pinus monticola* Near sea level	Oregon: Rhododendron Elevation 1700'
First appearance of aecia	1922	April 1	.....	.....	.....	.....	.....	.....
	1923	March 17	Early Apr.	Early May	.....	.....	.....	.....
	1924	March 15	" "	" "	.....	Early May	.....	.....
	1925	March 11	" "	" "	.....	.....	.....	.....
	1926	March 9	" "	Mid-Apr.	.....	.....	Late Feb.	.....
	1927	Mid-Mar.	Mid-Apr.	.....	Mid-May	.....	.....	.....
	1928	" "	Early Apr.	.....	Late Apr.	Early Apr.	Early Mar.	.....
	1929	.....	.....	Late Apr.	Early May	Late Apr.	Apr. 7	Apr. 6
	1930	.....	Late Apr.	.....	.....	.....	Mar. 16	Early Apr.
	1931	.....	Late Mar.	.....	.....	Early Apr.	Mar. 1	Mid-Mar.
	1932	.....	Mid-Apr.	.....	.....	.....	Apr. 5	Late Mar.
	1933	.....	Late Apr.	.....	.....	Mid-Apr.	Early Apr.	Early Apr.
	1934	.....	Mid-Mar.	.....	.....	.....	Late Feb.	Late Feb.
Main period of aecial sporulation	1922	Mid-Apr. to mid-Aug.	.....	.....	.....	.....	.....	.....
	1923	Early Apr. to early June	Mid Apr. to early June	Mid May to late June	.....	.....	.....	.....
	1924	Early Apr. to mid-June	Mid-Apr. to late June	" "	.....	Mid-May to mid-July	.....	.....
	1925	Late Mar. to mid-June	Late Apr. to late June	Early May to late June	.....	.....	.....	.....
	1926	Late Mar. to early May	Mid-Apr. to mid-May	Late Apr. to lat. May	.....	Mid-May to late May	.....	.....
	1927	Mid-Apr. to mid-June	Late Apr. to late May	.....	Early June to early Aug.*	Late Apr. to early June	.....	.....
	1928	Late Mar. to early May	Mid-Apr. to mid-June	.....	Mid-May to early July	Early May to mid-June	.....	.....
	1929	Late Apr. to late May	Late Apr. to mid-June	Early May to early June	Late May to early July	Mid-May to mid-June	Early May to mid-June	.....
	1930	.....	Early May to mid-June	.....	.....	Late Apr. to early June	Late Apr. to late June	Mid-Apr. to mid-May
	1931	Early Apr. to mid-May	" "	.....	.....	" "	.....	Late Mar. to early May
	1932	.....	.....	.....	.....	Early May to late May	.....	Late Apr. to late May
	1933	.....	.....	.....	.....	Late Apr. to early June	Late Apr. to late June	Late Apr. to early June
	1934	.....	.....	.....	.....	.....	.....	Mid-Mar. to mid-Apr.
Maximum aecial sporulation	1922	Late May	Late June	Mid-July	.....	.....	.....	.....
	1923	Early May	Mid-May	Early June	.....	Late May	.....	.....
	1924	Late Apr.	Early May	Late May	.....	Early June	.....	.....
	1925	" "	" "	" "	.....	.....	.....	.....
	1926	Early May	Late Apr.	Mid-May	.....	.....	.....	.....
	1927	Mid-May	Early May	.....	Mid-July*	Mid-May	.....	.....
	1928	Early May	Late Apr.	.....	Mid-June	Late May	.....	.....
	1929	" "	Mid-May	Late May	" "	" "	Late May	.....
	1930	.....	" "	.....	.....	Early May	Mid-May	Early May
	1931	Late Apr.	" "	.....	.....	" "	.....	" "
	1932	.....	Early May	.....	.....	Mid-May	.....	" "
	1933	.....	.....	.....	.....	" "	Mid-May	" "
	1934	.....	.....	.....	.....	.....	.....	Early Apr.
First pycnial drops	1922	June 24	.....	.....	.....	.....	.....	.....
	1924	Apr. 21	.....	.....	.....	.....	.....	.....
	1925	Mid-May	.....	.....	.....	.....	.....	.....
	1926	" "	.....	.....	.....	.....	.....	.....
	1927	Early June	.....	.....	.....	.....	.....	.....
	1928	.....	.....	.....	.....	.....	May 30	.....
	1929	.....	.....	.....	.....	.....	June 19	.....
	1930	.....	June 22	.....	.....	June 24	.....	.....
	1931	.....	Early June	.....	.....	June 25	.....	.....
	1933	.....	.....	.....	.....	July 1	.....	.....
	1934	.....	.....	.....	.....	.....	.....	Late May
Main period of pycnial production	1922	Late June to early Aug.	.....	.....	.....	.....	.....	.....
	1924	Mid-June to early Sept.	Early July to late Aug.	.....	.....	.....	.....	.....
	1925	Mid-June to Early Aug.	.....	.....	.....	.....	.....	.....
	1930	.....	Late June to late Aug.	.....	.....	.....	.....	.....
	1931	.....	" " "	.....	.....	Early July to late Aug.	.....	.....
	1933	.....	.....	.....	.....	" "	.....	.....
	1934	.....	.....	.....	.....	.....	.....	Early June to early Aug.
Last pycnial drops	1924	Sept. 16	Late Sept.	Mid-Aug.	.....	.....	.....	.....
	1926	.....	.....	.....	.....	.....	Aug. 12	.....
	1931	.....	Oct. 29	.....	.....	Oct. 29	.....	Oct. 7
	1934	.....	.....	.....	.....	.....	.....	Sept. 28

[a] An asterisk (*) following a date or period in the table indicates that the data were secured on *Pinus albicaulis;* all other data were secured on *P. monticola.*

TABLE 2.—*Dates of uredinial and telial development on ribes in different regions*

DEVELOPMENT on RIBES	Year	BRITISH COLUMBIA							UNITED STATES
		Coastal Region				Interior Region			
		Near sea level Elevation 0-200'	Garibaldi (Mile 22, PGE) Elevation 1100'	Mile 72 PGE Ry. Elevation 1300'	Prohibition Claim Plot and Tenquil Elevation 3000-5400'	Range of *Pinus monticola* Elevation 1150-2500'	Northern dry belt Elevation 1000-3600'	Southern dry belt Elevation 1000-3600'	Western Washington Elevation 0-500'
First uredinia	1922	May 18	.....	Mid-July	.....	.....	.....	.....	.....
	1923	May 7	May 21	Mid-June	.....	June 16	.....	.....	.....
	1924	May 21	Early June	Late June	.....	June 13	.....	.....	.....
	1925	May 10	May 10	May 23	.....	.....	.....	.....	.....
	1926	Apr. 25	Apr. 27	Early May	.....	Late May	.....	.....	.....
	1927	May 22	May 22	May 28	.....	.....	.....	.....	.....
	1928	May 11	.....	May 15	.....	June 5	.....	.....	May 11
	1929	.....	May 28	.....	June 17	Mid-June	.....	.....	May 22
	1930	May 8	Mid-May	.....	.....	Early June	.....	.....	May 6
	1931	.....	.....	.....	.....	Late May	.....	.....	.....
	1932	.....	.....	.....	.....	Early June	.....	.....	.....
	1933	.....	.....	.....	.....	June 13	.....	.....	.....
	1934	.....	.....	.....	.....	Early June	.....	.....	.....
First telia	1922	June 16	Early July	.....	.....	.....	.....	.....	.....
	1923	June 3	Mid-June	.....	.....	July 9	.....	.....	.....
	1924	Mid-June	" "	Early July	.....	Early July	.....	.....	.....
	1925	Late May	Late May	" "	.....	.....	.....	.....	.....
	1926	Mid-May	May 15	May 26	.....	.....	.....	.....	.....
	1927	June 7	June 2	June 17	.....	.....	.....	.....	.....
	1928	.....	May 30	.....	.....	.....	.....	.....	June 7
	1929	.....	Late June	.....	.....	.....	.....	.....	" "
	1930	.....	Mid-June	.....	.....	June 24	.....	.....	.....
	1931	.....	.....	.....	.....	June 11	.....	.....	.....
	1932	.....	.....	.....	.....	Late June	.....	.....	.....
	1933	.....	.....	.....	.....	July 6	.....	.....	.....
	1934	.....	.....	.....	.....	Late June	.....	.....	.....
Peak of telial production	1922	Late Sept.	Early Sept.	Early Oct.	.....	Early Sept.	.....	.....	Late Oct.
	1923	Late July	Late July	Mid-Aug.	.....	Mid-Aug.	.....	Late Sept.	Early Oct.
	1924	Late Aug.	Mid-Aug.	Early Sept.	.....	Early Sept.	.....	Late Oct.	October
	1925	Late Sept.	Late Sept.	Early Oct.	.....	" "	Early Oct.	Late Sept.	.....
	1926	Late Aug.	Late July	Early Sept.	.....	Late Sept.	Mid-Sept.	Early Sept.	Early Aug.
	1927	Mid-Sept.	Mid-Aug.	Late Sept.	.....	Early Sept.	Early Sept.	Mid-Sept.	Late Aug.
	1928	Early Sept.	Late July	.....	Mid-Sept.	.....	Late Aug.	Late Aug.	Mid-Aug.
	1929	Mid-Sept.	Late Sept.	.....	Early Oct.	Late Aug.	Early Sept.	Early Sept.	.....
	1930	Early Sept.	Late Aug.	.....	Mid-Sept.	Early Aug.	" "	" "	.....
	1931	Late Aug.	Mid-Aug.	.....	.....	Late July	.....	.....	.....
	1932	.....	.....	.....	.....	Early Aug.	.....	.....	.....
	1933	.....	.....	.....	.....	Late Aug.	.....	.....	.....
	1934	.....	.....	.....	.....	Late July	.....	.....	.....

interior region where white-bark pine, *Pinus albicaulis* Engelm., occurs, but some data were secured within its range in the higher elevations of the coastal region in the vicinity of the Prohibition Claim and Tenquil plots.

The dates given for stage of development of the rust on each host are based upon observations actually made at the respective places or within the regions listed. When the earliest and latest dates were noted on, or within a day or so of, the date of actual development, such dates are included. When observations of earliest and latest development were not made on actual dates but could be determined approximately from previous or subsequent observations made at the respective points, the dates are given for the part of the month in which the stage of development occurred. The months are divided into 3 periods: *i.e.*, early, to mean from the 1st to the 10th of the month; mid, to mean from the 11th to the 20th, and late, to mean from the 21st to the last day of the month, inclusive, respectively.

One of the earliest dates for aecial development was in the spring of 1934. Besides the data for that year presented in table 1, it may be mentioned here that in the vicinity of Fernwood, Idaho, aeciospore dispersal was observed March 10, and that it was estimated the first aecia made their

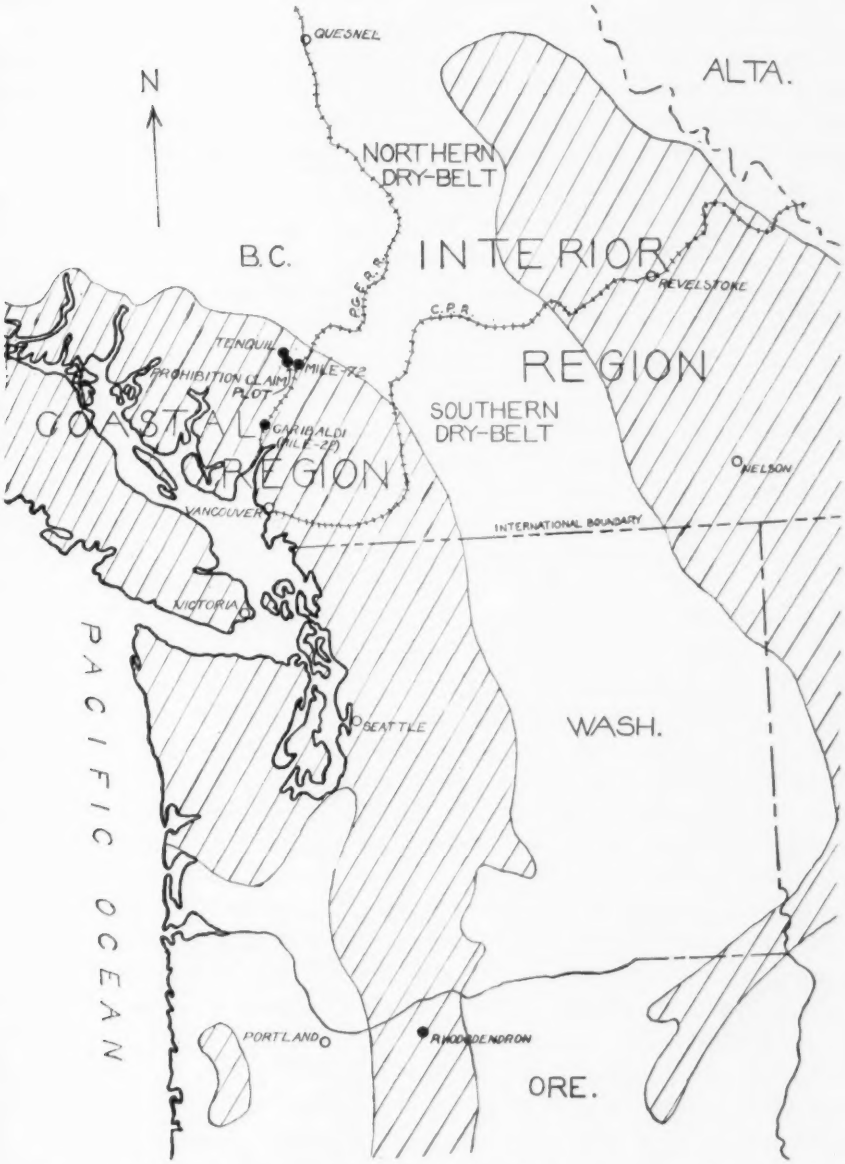

FIG. 1. Map showing regions and localities where data were obtained. Cross-hatched area outlines the botanical range of *Pinus monticola*. Solid black dots show localities referred to in tables 1 and 2.

appearance there in late February. Previous records for the State of Idaho have not been obtainable.

The data presented herein have been of value in analyzing the seasonal spread of the white pine blister rust in the Pacific Northwest. Lachmund[4] has shown the importance of time of aeciospore dispersal as a factor in influencing the degree and extent of seasonal spread of the rust.

DIVISION OF FOREST PATHOLOGY,
BUREAU OF PLANT INDUSTRY,
PORTLAND, OREGON.

[4] Lachmund, H. G. Seasonal development of ribes in relation to spread of *Cronartium ribicola* in the Pacific Northwest. Jour. Agr. Res. 49: 93–114. 1934.

SEED TRANSMISSION OF HELMINTHOSPORIUM OF CORN[1]

W. D. VALLEAU

(Accepted for publication February 27, 1935)

In the publication "Corn Diseases in Illinois"[2] the authors discuss helminthosporium leaf blight of corn (*Zea mays*) caused by the fungus *Helminthosporium turcicum* Pass., but make no mention of the possibility that the pathogen is seed-borne. In the discussion of the "Seasonal Cycle of Parasite" (page 108) they state: "Nothing is definitely known concerning the form or manner in which the fungus remains alive during the winter. It is assumed, however, that it overwinters on the old crop refuse, and that in the following season the spores are carried by wind or other agencies to the new corn crop." That *Helminthosporium* sp. is commonly present in corn seeds seems not to have been discovered by these writers in their extensive studies of seed infection. Likewise, Manns and Adams[3] do not list Helminthosporium as one of the organisms isolated from 7,205 seeds cultured, but list it amongst fungi found (p. 508) "in several instances . . . internal of seed corn."

In studies conducted by the writer between 1920 and 1925 on corn-seed infection, evidence was obtained that *Helminthosporium* sp. was present in seed of essentially all ears examined. Failure of others to observe this fact is due to the methods commonly used in studies of seed infection. In 1926 Valleau and Johnson[4] stated "In numerous tests conducted by the senior writer during the past five years, it has been determined that whereas the seeds of certain ears might appear to be free from fungi in germinator tests and culture dish tests conducted for a period of 6 or 8 days, yet when grown in sterile sand for longer periods, the presence of *F. moniliforme* and some others of the common seed-borne organisms could usually be demonstrated. *Helminthosporium* sp. is rarely found in germination tests of corn seeds or in cultures made from crushed seed, but if seedlings are grown in sand in an ordinary room and allowed to remain until they die, *Helminthosporium* sp. is quite commonly found fruiting on the stem and seed coat remnants." The object of the present note is to record the evidence for seed infection with *Helminthosporium* sp.

[1] The investigation reported in this paper is in connection with a project of the Kentucky Agricultural Experiment Station and is published by permission of the Director.

[2] Koehler, Benjamin, and James R. Holbert. Corn diseases in Illinois. Ill. Agr. Expt. Sta. Bul. 354. 1930.

[3] Manns, Thomas F., and J. F. Adams. Parasitic fungi internal of seed corn. Jour. Agr. Res. 23: 495–524. 1923.

[4] Valleau, W. D., and E. M. Johnson. A method of growing corn seedlings (*Zea mays*) free from seed-borne organisms. Jour. Agr. Res. 32: 1195–1198. 1926.

If corn seeds are planted in clean sand in a room with reduced light and kept at a temperature of about 80° F., germination is rapid and the second node or that from which the first permanent roots are derived is carried above the surface of the sand often as much as 3 inches. As a consequence, death of the seminal roots due to seed-borne organisms results in death of the plant; while in the field, by the time the seminal roots are destroyed, the permanent roots are well established. Death of seedlings occurs early or late depending perhaps upon the available food supply and upon the readiness with which seed-borne organisms are able to grow out from between the pericarp layers. This, in turn, appears to depend, to quite an extent at least, on the texture of the seed and the compactness of the pericarp layers. Seed-borne organisms develop readily from seeds that are starchy and have relatively loose pericarp layers. Seeds that are flinty, with closely compacted pericarp layers produce relatively long-lived seedlings in the sand box, and the fungi require a relatively longer time before they escape from the pericarp and attack the seminal roots and mesocotyl. Left a sufficiently long time under these conditions, nearly every corn seedling will die from the attack of seed-borne organisms. The longevity of sand-box seedlings from a given ear in comparison with that of seedlings from other ears, appears to be an inherited character and not an indication of the degree to which the seeds carry pathogenic organisms, as shown in table 1 where the seedlings of 3 "long-lived seedling" strains are compared with 3 "short-lived seedling" strains for 3 successive years, in self-pollinated lines of Boone County White.

TABLE 1.—*Length of life in the sand box, of 6 strains of Boone County White corn self-pollinated for 3 successive years.*

	Long-lived			Short-lived		
Strains No.	1	2	3	4	5	6
Selfed once	9.5	8.4	8.4	2.6	2.6	2.6
Selfed twice	9.0	9.7	11.1	1.4	2.1	1.9
Selfed three times	15.4	18.1	15.5	10.2	9.3	5.7

The longevity figure is the average number of days that seedlings lived in the sand box after death of the first plant. It will be noted that the short-lived strains were consistently short-lived through the 3 successive self-pollinated generations and the long-lived strains were consistently long-lived through the 3 self-pollinated generations. There was no evidence that length of life was associated with degree of infestation of the seeds with fungi.

Seeds from which the organisms grow readily, develop extensive dis-

colorations of the pericarp relatively early, while seeds from which the fungi grow slowly develop discolorations later and less extensively. The discolorations may be nearly completely prevented by treating the seeds with Bordeaux mixture, and may be aggravated by soaking the seed in 1 part of CaO to 4 parts of water for 20 hours. This treatment has a tendency to break down the seed coats and release the organisms. Seeds free from *Gibberella saubinetii* and *Diplodia zeae* develop various shades of red, bright green, dark olive, coal black, and a dirty brown. The various shades of red usually indicate the presence of *Fusarium moniliforme*. The cause of the bright green color has not been determined. Dark olive is commonly produced by *Alternaria* sp. and the black areas appear to be caused by *Helminthosporium* sp. A fungus that produces minute brownish sclerotia and develops a brown growth on potato agar was isolated from the dirty brown seed coats.

The discolorations appear to develop in areas of the seed coats already penetrated by the fungi before the seed is planted. Manns and Adams claimed that seed infection was largely limited to the area about the cap, but the writer has demonstrated by a study of serial paraffin sections that the fungi are distributed in the seed coats from one end of the seed to the other and are not at all limited to the portion near the point of attachment of the seed to the cob.

Seed infection evidently takes place early in the development of the embryo, as, in the extensive study of the time of infection of corn seeds, the writer found that ears harvested in late milk stage were as heavily infected, as indicated by seed coat discoloration and death of seedlings in the sand box, as seeds from matured and properly cured ears. From these studies and from studies of serial sections of corn seeds, it appears probable that infection occurs through the silks soon after pollination and that the fungi penetrate between the pericarp layers, in some cases developing sclerotium-like masses, before the pericarp walls have completed their development. It was not until many hundreds of seedlings had been grown in the sand box that it was realized that *Helminthosporium* sp. was a rather common seed-borne organism and that its presence was indicated by coal-black markings on the seed coats.

In a study of 500 ears that had been selected in the field and cured with heat, the smoothest and the roughest 50 ears were selected. From the smooth ears, 15 that produced the longest-lived plants in the sand box were selected and from the rough ears 15 that produced the shortest-lived plants. These were to be tested for yield in the field.[5] A study was then made of

[5] When tested in duplicate in the field, the increase in yield of smooth ears over rough ears was 39.43 per cent. This was attributed to physical characters of the plants and not to differences in seed infection. The pericarp layers are considered botanically

the organisms contained in the seeds of the 15 smooth ears, which would ordinarily have been considered the most nearly fungus-free. Twenty seeds from each ear were treated 3 minutes in 95 per cent alcohol; then 15 minutes in 1:1000 mercuric chloride solution, after which they were immediately planted in 2 series in sand that had been sterilized 35 minutes under 22 pounds' pressure. The seeds were set 1 inch apart in rows 2 inches apart. After planting, the sand was watered with sterile water and not watered again until near the end of the test when only a few plants remained. One series of plants was removed and examined between the 20th and 23rd days. If fruiting bodies were present on the seed coats these were recorded; if not, the seed coats were washed in sterile water and placed in a culture dish to be examined for fruiting bodies at the end of 24 or 48 hours. The other series was allowed to remain in the sand until the plants were dead, when the plants were removed and the fungi fruiting on the seed coats or mesocotyl determined by direct observation or by culturing.

Of 269 plants or seed remnants examined 94, or 35 per cent, had *Fusarium moniliforme* fruiting on them; 32 from 13 of the 15 ears, or 11.9 per cent, had *Helminthosporium* sp. fruiting abundantly either on the black areas on the seed coats or on the mesocotyl, often blackening it completely with a palisade of spores. On 11, Alternaria was fruiting; on 80 others, miscellaneous fungi were found. In the examinations it was found that *Helminothosporium* sp. was constantly associated with the coal-black areas and it, therefore, seems probable that black markings on the pericarp of seeds in the sand box are an indication of the presence of Helminthosporium.

In looking over earlier records on pericarp discoloration black markings were recorded on seed from 88 of 109 ears of Boone County White and from 73 of 76 ears of Reid's Yellow Dent. A total of 28.5 per cent of 2058 seeds of Boone County White and 47.4 per cent of 1069 seeds of Reid's Yellow Dent developed black markings, suggesting the presence of Helminthosporium.

It appears from these studies and from test-tube and culture-plate studies, that organisms between the pericarp layers are often sealed and not capable of growing until the pericarp walls are broken down by other processes than those occurring during germination under aseptic conditions. It seems evident that the usual culture-plate method cannot be relied upon for the accurate determination of organisms carried by seed.

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to be a leaf folded about the ovary. Therefore, long, deep-dented seeds should be borne on large-leaf plants inefficient in the use of water, and small, flinty seeds on plants with relatively smaller leaves, more efficient in the use of water.

PHYTOPATHOLOGICAL NOTES

Some Factors Affecting Infection With and Spread of Diplodia zeae in the Host Tissue.—A better knowledge of factors affecting the infection and subsequent development of *Diplodia zeae* (Schw.) Lév. in the tissues of corn, *Zea mays* L., is important in connection with any study dealing with the cause of resistance to this fungus.

The following observations, though detached and fragmentary, may prove suggestive to those interested in the study of this fungus and its host relationships. They are presented as a possible lead for future investigations of a disease that has yielded relatively few tangible leads in the past.

In general, stalk tissues become more predisposed to *Diplodia zeae* as the grain approaches maturity. Conditions that predispose stalk tissues to *D. zeae* and those that predispose the ear and grain to the fungus are not always the same. Moreover, resistance of one set of tissues of the corn plant to *D. zeae* does not insure resistance of other parts of the same plant.

In 1931 stalk tissues of certain inbred lines not permitted to set grain were invaded less rapidly and to a less extent following hypodermic injection of a spore suspension of *Diplodia zeae* than were stalk tissues of comparable plants of the same inbreds allowed to fruit normally. The inoculations were made in the pith tissues of the third or fourth internodes above the soil line 3 weeks after the pollination period.

The stalk tissues of plants whose leaves were injured by bacterial leaf blight the latter half of August, during the epidemic of that disease in field corn in 1932, were found to be invaded by Diplodia, prior to maturity of the grain, to a significantly greater extent than were the stalk tissues of comparable plants only small areas of whose leaves had been killed by bacterial leaf blight.

Artificial defoliation experiments have been conducted from 1932 to 1934, inclusive. During the last 10 days in August, from 25 to 35 per cent of the tip end of each leaf of part of the plants was removed by clipping. Stalk tissues of these partly defoliated plants were invaded earlier and to a much greater extent by *Diplodia zeae,* following both natural infection and hypodermic injection of a spore suspension while the leaves were still green, than were stalk tissues of comparable plants none of whose leaves were clipped. A summary of the results for the 3-year period is given in figure 1.

Infestations of second-brood chinch bugs, which feed very largely on the carbohydrates they remove from the lower parts of the corn plant, have been found to be followed by a marked increase in the rate at which *Diplodia zeae* invades the stalk tissues prior to maturity, excepting in strains highly resistant to injury from the attack of second-brood chinch bugs. During 1933

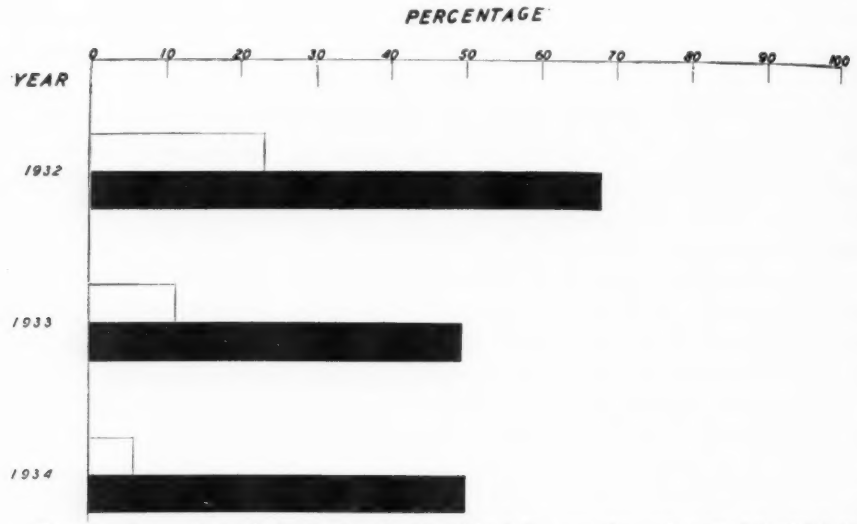

FIG. 1. Percentage of plants at maturity with stalks conspicuously rotted by Diplodia in plant populations whose leaves were not clipped (white bar) and in plant populations whose leaves were partly clipped (black bar), Bloomington, Illinois, 1932–1934, inclusive.

and 1934 the amount of grain damaged by *D. zeae* was almost always very much greater where the corn plants were infested by second-brood chinch bugs, but not killed prior to maturity, than where comparable plants were not infested with second-brood chinch bugs.

Johnson and Christensen[1] have reported that ears borne on smut-infected plants were more likely to be damaged by ear rots than ears borne on smut-free plants.

Stalk and shank tissues, and also the ears, of plants exposed to both naturally occurring and artificially produced chilling and freezing temperatures, as they approach complete maturation, usually have been invaded more rapidly and to a much greater extent by *Diplodia zeae* than have the same tissues of comparable plants not exposed to such temperatures.

In each case cited, increased susceptibility of the stalks to the fungus is associated with conditions that may well result in a reduction of the carbohydrate reserves of the plants. Apparently this situation does not hold, however, for susceptibility in the ears.—J. R. HOLBERT, P. E. HOPPE, and A. L. SMITH (Cooperative investigations between the Division of Cereal Crops and Diseases, Bureau of Plant Industry, U. S. Department of Agriculture, and the Illinois and Wisconsin agricultural experiment stations).

[1] Johnson, I. J., and J. J. Christensen. Relation between number, size and location of smut infections to reduction in yield of corn. Phytopathology 25: 223–233. 1935.

A Top-necrosis Virus Found in Some Apparently "Healthy" Potatoes.[1]—Murphy and McKay[2] found in their study on a comparison of some European and American diseases of the potato that they had considerable difficulty in introducing certain American viroses into healthy President plants. There was found to be throughout the American material a latent virus or viruses, which had a severe necrotic effect on President. They found that this latent virus (or more probably viruses) was present in the "healthy" Green Mountain, as well as in the obviously diseased plants.

The writer has made grafts of mild mosaic, crinkle mosaic, and leaf-rolling mosaic from Earliest of All and Bliss Triumph on healthy Arran Victory and President. In many cases top necrosis developed on these two varieties. A small number of grafts of scions from "healthy" Burbank and Earliest of All were made to President, and some of these plants developed a top necrosis. These symptoms could not be ascribed to the so-called latent mosaic of X-virus, which is universally present in healthy American commercial potatoes, since that virus produces in these two varieties only a mosaic type of mottling without necrosis.

In 1933 the writer received several diseased and healthy tubers of different European varieties from R. N. Salaman of England. Among the tubers was the variety Up-to-Date, which is a symptomless carrier of virus B. Bawden, according to Smith and Brooks,[3] states that the streak-producing B-virus is almost universal in this variety, and is usually accompanied by virus X, (the latent mosaic of "healthy" commercial potatoes). Grafts from Up-to-Date to Arran Victory and President produced a top necrosis.

In order to determine whether or not the X-virus is required to produce this disease, grafts of Up-to-Date were made to the U. S. D. A. Seedling No. 41956, which is resistant to X-virus.[4] By this procedure it is possible to remove the X-virus and to retain the B-component. Grafts of the infected seedling to Arran Victory produced typical top necrosis symptoms. Grafts of the infected seedlings to tomato transferred the B-virus, but failed to develop any noticeable symptoms. The presence of the B-virus was demonstrated when grafts of the infected tomato plants produced top necrosis in Arran Victory and President.

[1] This paper is based upon investigations carried on as a cooperative project between the Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, U. S. Department of Agriculture, and the Oregon Experiment Station at Corvallis, Oregon.

[2] Murphy, Paul A., and R. McKay. A comparison of some European and American virus diseases of the potato. Sci. Proc. Roy. Dubl. Soc. 20: 347–358. 1932.

[3] Smith, K. M. (Foreword by F. T. Brooks). Recent advances in the study of plant viruses, 423 pp. P. Blakiston's Sons. Philadelphia, 1934. (See page 316).

[4] Schultz, E. S., and W. P. Raleigh. Resistance of potato to latent mosaic (Abstr.) Phytopathology 23: 32. 1933.

Several grafts of Up-to-Date were made to the following American varieties: Burbank, White Rose, Irish Cobbler, Earliest of All, and Bliss Triumph. Symptoms failed to develop either during the current season or in the progeny. These results indicate that the American varieties tested probably serve as symptomless carriers of virus B, although return grafts from grafted American varieties to President or Arran Victory have not yet been made, because of insufficient plants of these varieties.

Since the American varieties tested fail to develop symptoms when grafted with virus B, and since this virus produces top necrosis in Arran Victory and President, it is believed that the virus component found in apparently healthy tubers of some American varieties in addition to virus X, is the same as Bawden's virus B.

Preliminary results indicate that not all varieties of American potatoes carry this virus, since in some cases a mottling without top necrosis resulted when grafts of scions of apparently healthy American potatoes were made on Arran Victory or President. Plans are being made to test a large number of plants of American varieties for the presence of this top-necrosis virus. Although European workers have referred to the presence of virus B in American varieties, this fact has not previously been confirmed by American investigators. This is undoubtedly due to the fact that most, if not all, or our American varieties are symptomless carriers of this virus and, since it fails to produce any apparent symptoms on tobacco or Datura, it is difficult to detect. When it is introduced into European varieties, like President, Arran Victory, or Epicure, the presence of this virus can be readily demonstrated. Further studies of this virus component are in progress.—T. P. DYKSTRA, *Oregon State College*, Corvallis, Oregon.

An Apple Canker Caused by Monochaetia mali.—An apple canker caused by *Monochaetia mali* (E. and E.) Sacc. (fig. 1) has been occasionally seen by the writer since 1910. In May, 1911, successful inoculations were made using spores from pure cultures, and in October of the same year the fungus was reisolated from the resulting typical cankers. *Monochaetia mali* fruited copiously on cankers produced from artificial inoculations, as it does on those occurring naturally. The fungus enters through deep wounds, grows deeply into the wood, and subsequently attacks the resulting wound callus, producing numerous fruiting bodies on both the exposed wood and the layer of wound callus. The killing of successive layers of wound callus (Fig. 1) results in the formation of a type of canker somewhat resembling the European apple canker caused by *Nectria galligena* Bres. Since the canker caused by *Monochaetia mali* appears to be uncommon, it cannot be considered as a serious apple disease.—JOHN W. ROBERTS, Division of Fruit and Vege-

FIG. 1. An apple branch in two pieces, showing a canker caused by *Monochaetia mali*. The canker on the smaller piece is a continuation of that on the larger piece. Note killing of successive callus layers, spread of canker, and presence of fruiting bodies. Collected by Leslie Pierce, Vincennes, Ind., June, 1934. ×1.

table Crops and Diseases, Bureau of Plant Industry, U. S. Department of Agriculture.

Notes on the Occurrence of Tuberculina maxima on the Aecia of Cronartium cerebrum.—In a recent article Hubert[1] listed *Cronartium cerebrum*

[1] Hubert, E. E. Observations on *Tuberculina maxima*, a parasite of *Cronartium ribicola*. Phytopath. 25: 253–261. 1935.

(Peck) Hedgc. and Long as one of the hosts of *Tuberculina maxima* Rostrup. In the collections of the Division of Forest Pathology, Bureau of Plant Industry, Washington, D. C., there are 225 naturally infected specimens of *C. cerebrum* occurring on 14 species of Pinus in the eastern United States. Only 7 of these specimens are infected with *T. maxima*. All 7 are on *P. virginiana* from Maryland, Virginia, and Pennsylvania. The above-mentioned specimens of the rust, a large number of which were collected by the writer, are typical of thousands of *C. cerebrum* galls that the writer has observed during the past 30 years. Since *T. maxima* was collected so infrequently, it must be concluded that it is rare and of no economic importance on *C. cerebrum*.—GEO. G. HEDGCOCK, *Division of Forest Pathology, Bureau of Plant Industry,* Washington, D. C.

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Supplement to

Phytopathology

Volume 25 Number 5

May, 1935

PUBLISHED MONTHLY BY THE SOCIETY

OFFICE OF PUBLICATION

COR. LIME AND GREEN STREETS, LANCASTER, PA.

Entered at the post-office in Lancaster, Pa., as second class matter.

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NINMAN, H. J.	1916	1934
PEIRCE, HAROLD	1913	1933
SEYMOUR, A. B.	1919	1933
STEVENS, F. L. (charter)	1908	1934

* Members who have died since the publication of the list of members in September, 1932.